Registered at the G.P.O., Sydney, for Transmission by Post as a Newspaper. Published Weekly. Price 1s.

THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—9TH YEAR. SYDNEY: SATURDAY, MARCH 25, 1922. No. 12.

THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—9TH YEAR. SYDNEY: SATURDAY, MARCH 25, 1922. No. 12.

Table of Contents

THE IMPORTANCE OF THE ACID CONTENT OF THE GASTRIC JUICE IN THE ÆTIOLOGY, SYMPTOMATOLOGY AND TREATMENT OF GASTRIC AND DUODENAL ULCERS.[1]

BY A. E. MILLS, M.B. (SYDNEY),
Professor of Medicine, University of Sydney.

I HAVE chosen this subject, firstly, because I feel sure it is one that will be of interest to all practitioners and, secondly, because I think insufficient attention is paid to the physiological and pathological aspects of disorders of the stomach.

Of recent years considerable advance in our knowledge of the causation, symptoms and treatment of ulcers of the stomach and duodenum has been made. This address is to be looked upon chiefly as a very brief review of some of the work done by men like Bolton, Hurst, Moynihan, Sippy and the Mayo brothers, men who have devoted themselves particularly to the consideration of these disorders.

Primary Factors in the Ætiology of Gastric and Duodenal Ulcers.

Bolton, in his excellent book, "Ulcers of the Stomach," describes the effect of a substance which he calls gastro-toxin. It is produced in the serum of a rabbit immunized with the cells of the mucous membrane of a guinea-pig. When he injected some of the serum of the rabbit thus immunized into the peritoneal cavity of a guinea-pig, necrosis of some and damage to other cells of the mucous membrane of the guinea-pig's stomach resulted. This effect was invariable. Following upon the necrosis an ulcer or ulcers formed. But for us just now this is the important point: if the influence of the gastric juice was eliminated by neutralization of the acid, the necrosed area healed; no ulcer resulted.

With regard to the ætiology of gastric and duodenal ulcers, the question we may straightway ask ourselves is: "Why do ulcers more frequently form in the stomach and duodenum than in other parts of the intestinal tract?" It is not easy to give the answer to this question. Some important facts must be borne in mind. Firstly, the stomach and duodenum are bathed with an acid secretion and I will endeavour to bring forward evidence to show that this acid secretion plays a very big part in the formation of the ulcers and in the prevention of their healing and, further, that the treatment, whether it be surgical or whether it be medical, depends for its success upon neutralizing the acidity of the gastric secretion or minimizing the time during which the acid juice bathes the ulcer.

[1] Being an introductory address delivered at the first Post-Graduate Course held in Sydney on January 9, 1922.

There are numerous cases on record to show that the poisons absorbed after extensive burns produce necrosis of the cells of the mucous membrane of the stomach and duodenum—more frequently of the duodenum than of the stomach—and that these necrotic areas rapidly become ulcers, owing to the action of the gastric juice. Ponfick, cited by Bolton, has recorded a case in which lesions of the stomach and duodenum were found eighteen hours after a burn. There was no doubt these lesions were directly the result of the burn and not merely of a secondary bacterial infection, seeing they occurred so early in the course of the disease. Further, poisonous substances obtained from infusions of burned tissue or blood from burned animals produced similar phenomena. These and other experiments show that poisons of metabolic origin formed as a result of cellular destruction are able so to damage the cells of the gastric mucous membrane than an acute ulcer may result. Further, it is also well known that in some cases of uræmia ulcers have formed in the duodenum and other parts of the intestines and indeed death has resulted from hæmorrhage due to an ulcer having eroded a large vessel.

Again, ulcers of the stomach and duodenum have been observed in septicæmic states. The introduction of pus intravenously produced ulcers.

Rosenau found in animal experiments that streptococci, irrespective of their source, if of sufficient virulence, exhibited an affinity for the gastric mucous membrane. When injected intravenously ulcers of the stomach and duodenum resulted.

Acid Gastric Secretion the Determining Factor.

All the evidence then seems to show that if the walls of the stomach or duodenum are sufficiently damaged by blood poisons of metabolic origin (gastro-toxin) of cellular disintegration (burns) or bacteria, digestion of the damaged cells by the gastric juice takes place and an ulcer results. The particular fact I wish to insist upon is the important part, even though it is not the primary part, that the acid gastric juice plays in the production of ulcers of the stomach and duodenum.

It would seem then that products of cellular degradation or micro-organisms have the power of damaging some of the cells of the stomach or duodenum and that this probably is the first stage in the formation of an ulcer. This being granted, the question immediately arises: "When these products are circulating in the blood, why should these ulcers form in the stomach and duodenum more frequently than in other parts of the digestive tract?" To me it seems highly probable that the reason for this increased frequency is to be found in the fact that these damaged cells, not necessarily necrosed, are now no longer able to resist the disintegrating action of the acid gastric juice; in other words, that they become digested, a solution of continuity takes place and an ulcerated surface results. The poisons or bacteria do not cause the ulcer—they damage or produce malnutrition or necrosis of the cells; the actual ulcer results from the digestion of the damaged cells by the acid juice.

The Significance of Pain.

Secondly, with regard to symptomatology, I wish particularly to draw your attention to the fact that in ulcers of the stomach and duodenum the pain or discomfort or distress, which is the chief indication for our recognition of these disorders, is not due, as is still sometimes held, to the contact of the ulcer with food particles or directly due to the ulcer being laved with the acid digestive juice. There is abundant evidence to show that these ulcers may be mechanically irritated in various ways without giving rise to any pain and many experiments have been undertaken to show that contact of the ulcer with a juice of a concentration greater than that ever found in the stomach of man does not produce pain.

Now it is well known that in cases of ulcer of the stomach and duodenum there are periods when there is neither pain, discomfort nor distress, followed by periods when pain or discomfort or distress is marked. We surely cannot entertain the belief that during these periods of cessation of distress the ulcer has healed. What, then, is the cause of the pain?

I think we must look for the explanation of the pain to a common cause; and that common cause is tension. It is a well-known fact that increased tension in the gall bladder or urinary bladder causes great pain. But tension must not be confused with distension, for distension may occur without producing increased tension. This statement, however, may be accepted—that wherever there is a sufficient increase of intra-visceral tension pain results. And, indeed, in the solid viscera the same principle obtains; witness the great pain and tenderness that results from a large infarct of the spleen or kidney and note the painful and tender liver when there is great tension within its capsule due to acute congestion following cardiac failure or from a rapidly growing malignant tumour.

But probably the best example of the painful effects of increased tension is to be found in narrow tubes down which foreign bodies are being forced. Let me remind you of the unbearable pain which results from the passage of a stone down the ureter or the passing of a stone down one of the bile ducts.

How, then, can we bring this common cause for visceral pain to explain the symptoms of discomfort that are so commonly found in ulcerated states of the stomach and duodenum? Let it be borne in mind that in many cases of gastric neurosis, found so commonly in people who are run down in health and who have lost weight and have had much mental worry, there is distress which simulates very closely the distress experienced in frank cases of gastric or duodenal ulcer. In these cases we have a condition of hyperchlorhydria, just as is found, as a rule, in gastric and duodenal ulcer.

May I be permitted to quote from an article entitled "The Symptomatology of the Disorders of the Stomach from the Physiological Aspect," published in THE MEDICAL JOURNAL OF AUSTRALIA, October 5 and 12, 1918:

> Accepting the view of the acid control of the pylorus through the myenteric reflex, we can readily see that this reflex may, like other reflexes, be excited or depressed. If acid be the adequate stimulus, as seems to be the case,

then hyperacidity, as we find clinically, should still further excite it. We have some evidence for this, for in cases of duodenal ulcer or hyperacidity there is hypermotility. Again, from an ulcerated surface the afferent stimuli for the reflex might conceivably be very much stronger than from a sound surface. This would lead to a more pronounced end result. The strongly marked spasm of the circular fibres, leading to a well marked incisura in a case of gastric ulcer, suggests increased stimulus and increased end effect. The stimulus arising from a duodenal ulcer should also increase reflex activity. The end result should be marked. That this is so is proved by the strengthened peristalsis and pyloro-spasm. Now, where there is pyloro-spasm there is greatly increased tension; where tension is greatly increased pain is experienced. The cause of the gastric pain, then, in appendicitis, gall bladder disease, duodenal ulcer, etc., is increased tension, which must result from the pyloro-spasm.

If pyloro-spasm be not accepted as the complete explanation for the causation of pain experienced in gastric and duodenal ulcer, there can be little doubt that the acidity of the gastric juice plays an important part in the production of the pain, for we have only to remember, as we will see when we come to the treatment, that neutralization of the acidity by alkalies or diminishing its concentration by food abolishes the pain as a rule within a few minutes.

Leaving the question of the causation of pain, there are some other aspects of the pain of gastric and duodenal ulcer on which I would like to dwell. When the stomach is empty of food pain is absent, but I would have you remember that five to fifteen cubic centimetres of fluid may be found in a fasting stomach, though the acidity of this fluid is not very high. If when the stomach is empty there is no pain, the distress must be due to the stomach contents. Now all the evidence goes to show that the distress is not due to the solid particles of food, unless, perchance, they be of great size, but that the distress is due to the fluid contents. This is shown by the fact that the distress is greatest in the night in those cases of hypersecretion, when there may be from one hundred to five hundred cubic centimetres of fluid in the stomach, with only small fragments of food. It has also been shown that the higher the degree of free acidity present, the greater the distress.

The following clinical facts relating to the distress of gastric and duodenal ulcer, cited by Sippy in one of his articles on "Ulcers of the Stomach and Duodenum," are of great importance:

(*i.*) The distress associated with ulcer is absent when the stomach is empty.
(*ii.*) It appears an appreciable time after eating.
(*iii.*) It is relieved by food or drink or by vomiting.
(*iv.*) It is relieved by alkalies.
(*v.*) The distress is associated with an adequate free hydrochloric acid content of the stomach juice.
(*vi.*) The distress is associated with an increased amount of gastric juice.

If in suspected cases of ulcer these conditions are not present, it is very probable that an ulcer does not exist.

We meet with certain patients who complain of pain in the morning before breakfast. In these cases the probability is that, if there is no obstruction caused by the ulcer, the pain is not due to the ulcer. It is generally associated with neurasthenia. The taking of food relieves the pain by lessening the acidity of the juice and the adequate stimulus for contractions of the stomach are then wanting.

But freedom from pain is more quickly and more completely brought about by the administration of alkalies which neutralize the gastric juice. Indeed, we can go further and say that in all probability the distress experienced in disorders of the stomach is not due to an ulcer, if, with the stomach empty, 0.12 gramme of calcined magnesia and 0.12 gramme of bicarbonate of soda do not afford relief. The administration of such an alkaline powder generally gives relief in five to ten minutes, though in some cases a longer time is required. If the relief is not complete after the administration of another powder in twenty minutes' time, then in all probability the distress is not due to an ulcer and another cause for the pain must be sought.

We meet with patients whose discomfort or distress from the ulcer shows definite periods of improvement, followed by periods of retrogression; this periodicity has gone on for ten, fifteen or twenty years. Many writers have laid stress on this periodicity in the attacks of discomfort. Probably in these cases the ulcer heals to a certain extent and then breaks down again.

The distress of an ulcer usually appears from one to three hours after eating. It very seldom appears, unless there be complications, as late as five hours or as early as half an hour after the taking of food. The distress does not worry the patient while he is eating or immediately after eating, but it does appear when the food is well saturated with hydrochloric acid, when, in fact, there is much free acid present. All these statements show that the pain is closely related with the presence of acid; but it must be borne in mind that there are other conditions in which there is an increase of acid, that is to say, hyperchlorhydria, which mimic in their symptomatology those of gastric and duodenal ulcer. Such conditions are gall stones, chronic appendicitis and certain neurotic states. The differentiation of these disorders from that of ulcer is not always easy, but reliance upon those guiding principles which I have previously mentioned, will generally keep us in the right path. In these disorders you will notice that the distress, which is the chief symptom, is closely associated with increase of acidity. Gall stone disease seems to me to offer the greatest difficulty in differentiation, but the pain of ulcer is never so severe as that of gall stones, although it must be admitted that gall stones may exist for a long time and produce gastric disorders without exhibiting the classical symptoms of severe gall stone colic. One other point may be mentioned; with ulcer the pain is experienced day after day and after each meal; with gall stone colic the attacks are never so frequent and never so regular. The symptoms of indigestion that are experienced in gall stone disease, chronic appendicitis and neurosis depend upon the acidity of the gastric juice and they also are relieved by alkalies.

It must now be apparent what an influence the

acidity of the gastric juice plays in the production of gastric pain or distress.

Dealing as I am in this address chiefly with the part played by the acidity of the gastric juice, I do not intend to refer to the great assistance given by X-rays and test meals in the diagnosis of ulcers of the stomach and duodenum. Although I admit that X-rays may and do at times give unequivocal evidence of the presence of ulcer in the stomach and duodenum, still the evidence given by X-rays must always be taken in conjunction with the facts elicited by careful inquiry into the history of the patient.

Treatment.

We turn now to the question of treatment. I certainly cannot go as far as Hurst when he says that "the surgical treatment of gastric and duodenal ulcer is a confession of failure"; but I think that it will be admitted that the routine operation of gastro-enterostomy for ulcer or suspected ulcer is to be strong deprecated. I think we may go further and agree with Moynihan when he says: "I am personally adverse to a gastro-enterostomy, except in cases of existing or threatened obstruction, that is, in cases of duodenal ulcer or in special circumstance only of pre-pyloric ulcer." There is abundant evidence that surgical treatment for ulcer of the stomach and duodenum gives immediate and in some cases permanent relief of all symptoms of distress. Such a result can hardly be classed as a failure, but I feel very strongly that medical treatment, properly carried out, will frequently obviate the necessity for surgical interference. I am confirmed in this view by the definite statement of Moynihan, for he says: "A really serious attempt to treat all cases of gastric ulcer by medical means should be made." And: "It is at least arguable that the necessity for surgical relief in many patients is due to a too perfunctory trial of medical treatment in the earlier attacks." I will attempt to show later that medical treatment is not to be restricted to the earlier attacks, that even in cases of advanced ulcers associated with pyloric obstruction due to inflammatory thickening around the ulcer, medical treatment, if properly carried out, can yield just as brilliant results as those obtained by surgical treatment.

The first question we have to ask ourselves is: "Why does gastro-enterostomy in cases of ulcer give such beneficial results?" This leads me to put before you a further question: "Seeing that ulcers in other parts of the alimentary tract, when brought under suitable conditions, heal readily enough, why should not ulcers in the stomach and duodenum heal in a similar manner?" Why, for example, do typhoid ulcers heal? We do not speak of a chronic typhoid ulcer, but how common it is to find evidence of a chronic gastric or duodenal ulcer! In seeking for an explanation of the failure on the part of the body to bring about a healing of a gastric or duodenal ulcer, we may well ask ourselves: "Are these ulcers subjected to different conditions or influences? And, if so, what are these different conditions or influences?" The only answer to these questions, and I think it is a quite sufficient one, is that they are subjected to different conditions, namely, that these ulcers are subjected for many hours a day to the action of an acid digestive juice.

This fact, I think, explains the reason why a gastro-enterostomy often gives such relief and has such beneficial results, for it allows the stomach contents to escape rapidly and thereby limits very materially the time during which the acid gastric juice comes in contact with the ulcer. If the ulcer is laved with an irritating juice for nine or twelve or fifteen hours a day and if, as a result of surgical treatment, the conditions are so altered that the time is lessened by half or even more, then the ulcer is placed in a much better condition for healing. In other words, the ulcer is placed in a condition of comparative rest; in the treatment of such conditions rest is a fundamental principle.

It affords, I think, an explanation of those cases which my surgical colleagues have related to me—cases of pyloric obstruction associated with ulceration deemed to be malignant for which they have performed a preliminary operation of gastro-enterostomy. Two or three weeks later, having opened the abdomen with a view of excising the ulcerated portion, they noticed that all traces of the ulcer and obstruction had disappeared. That is to say, the ulcer, having been removed from the corrosive influence of the acid juice, rapidly healed.

It must not be forgotten, also, that ulcers do heal in the absence of either surgical or medical treatment. The scars that are found *post mortem* bear indubitable testimony to this. Truly the recuperative powers of the body are marvellous in the extreme.

If you agree with me, then, that the greatest factor in the retardation of the healing of an ulcer is the corrosive action of the acid gastric juice, the guiding principle in all treatment should be the elimination of this factor. And it should also follow that if this corrosive and irritating influence is removed, the ulcer of the stomach or duodenum should readily heal. We need not concern ourselves with the digestive ferment, pepsin, for all we have to do is to aim at the neutralization of the acid content of the gastric juice. Pepsin acts only in the presence of an acid and is inert in an alkaline medium.

This is the basis of the medical treatment advocated by Bernard Sippy, of which Moynihan says: "The most rational method is that introduced by Sippy, which would appear to meet more combatantly those conditions in the stomach which we believe must be controlled before the ulcer can have a chance to heal."

I do not think I can do better than summarize Sippy's method of treatment. Let me repeat that the treatment consists essentially in maintaining an accurate neutralization of all free hydrochloric acid during the time the food and its accompanying secretion are present in the stomach. When the diagnosis of gastric or duodenal ulcer has been made, the patient should be put to bed for about three weeks. Then he may be allowed up for gentle exercise. If there be complications such as hæmorrhage or peritonitis, the rest in bed should be longer. If there be no complications, the patient may be

allowed to go about and do all or at any rate part of his usual work after four or five weeks. Sippy, in his latest article, does not advocate preliminary starvation, for he maintains that when the acidity of the juice is neutralized, starvation serves no useful purpose. He recommends that ninety cubic centimetres (three ounces) of milk and cream in equal parts be given every hour from 7 a.m. to 7 p.m. for the first two or three days and afterwards an egg or bread and butter or biscuit may be added to one of the morning feedings and that ninety cubic centimetres (three ounces) of a milk food, such as oatmeal or farina, may be added to one of the afternoon feedings. The quantities of egg and cereal food are gradually increased, until at the end of the first week the patient may be taking daily ninety cubic centimetres of milk and cream every hour from 7 a.m. to 7 p.m., together with two or three soft boiled eggs (one at a time) and 180 or 270 cubic centimetres (six or nine ounces) of a cereal (ninety cubic centimetres at a time).

Custards, cream soups, vegetable *purée* and other soft foods may be substituted now and then for the milk and cream feedings. Jellies and marmalades may be added if desired. The quantity of food should be sufficient to cause a gain of 0.9 or 1.4 kilograms a week. This method of feeding tends to neutralize the free acidity of the juice; but to control the acidity effectually Sippy recommends that an alkaline powder, consisting of 0.6 gramme of calcined magnesia, 0.6 gramme of sodium bicarbonate, alternating with a powder containing 0.6 gramme of calcium carbonate and 1.8 grammes of sodium bicarbonate, be taken midway between the feedings. In addition, after the last feeding four or five doses of the powders should be given every half hour. It is well to remember, as Sippy points out, that calcium magnesia and calcium carbonate have respectively four and two and a half times the neutralizing power of sodium bicarbonate. Although the acidity is controlled by hourly feedings and hourly administration of the alkali, the acidity may also be controlled by feeding every two or three or four hours or even by feeding three times a day if adequate doses of alkali be given at sufficiently frequent intervals; but the plan of feeding every hour and giving the alkali between the feedings for the first four or five weeks is the method specially recommended by Sippy.

The powder may be stirred up with water immediately before it is taken. Sometimes the magnesia produces a little diarrhœa. The other powder mentioned should then be substituted for the magnesia and soda. On the other hand, if there be a tendency to constipation, more of the powders consisting of magnesia and soda, in place of the lime and soda, should be taken. A slight tendency to looseness is to be preferred. In order to determine whether the acidity is being controlled aspiration of the stomach contents should be performed on two afternoons and three evenings each week during the time the patient is under careful control. The evening aspiration should be done half an hour after the last powder. If it be found that the acidity is not sufficiently controlled, 0.3 gramme of calcium carbonate should be added to each powder. Even though there should be a little secretion containing free acid after the last powder has been administered, it is not likely to have very much influence in preventing the healing of the ulcer. It will act only for a very little time—nothing comparable to the time during which the ulcer may be subjected to the action of the juice following even gastro-enterostomy.

After the fourth week the routine of feeding may be changed to three meals daily, the milk and powders being still taken at hourly intervals. The total bulk of food should not exceed 300 to 450 cubic centimetres. Tea and coffee may now be allowed and later cooked fruit, later still raw fruit—a wide variety—may be added.

If desired, later on the patient may occasionally take an ordinary full meal and later still may take three ordinary full meals a day, but after a full meal one powder each half hour for six doses, or two powders at the end of each hour for three doses should be taken. It is Sippy's custom to cease giving powders for five days at the end of ten weeks and then to resume them as before for five or six weeks. This plan is to be followed until the treatment is discontinued, but during the period when no powders are taken, the frequent feedings should be resumed. Sippy recommends that treatment should be continued for a year.

Hitherto we have considered simply the medical treatment of gastric and duodenal ulcer without complications. What have we to say with regard to the medical treatment of an ulcer producing more or less pyloric obstruction? Well, the medical treatment, according to Sippy, is the same in principle, that is to say, the aim should be to keep the acid neutralized, but slight alteration in the method of the treatment is required, on account of the fact that in these cases there is generally found an excessive secretion of a highly acid gastric juice after the food has passed through the more or less obstructed pylorus and this secretion, unless removed, will have an undesirable corrosive action on the ulcer. Firstly, it may be necessary to give a larger quantity of alkali to control the acid and, secondly, it is found that the best results are obtained by emptying the stomach every night with the tube, half an hour after the last powder is taken.

Sometimes it has been found necessary to give 1.8 grammes each of calcium carbonate and sodium bicarbonate every hour between the feedings and every half hour after each feeding until 9 p.m.. It may even be found necessary to aspirate the stomach twice in the evening after the last meal, say, at 9.30 and 11.30 p.m., to get rid of the excessive secretion. If there be considerable secretion at 9.30 p.m. powders should be given every half hour until the stomach is again aspirated at 11.30 p.m.. It has been found that in these cases under this treatment after a few days the excessive secretion has diminished to fifteen or twenty cubic centimetres of fluid. This is practically the normal amount found in a fasting stomach.

May I be permitted to read you the claims made by Sippy as to the beneficial effects which he has obtained by this method of medical treatment, treat-

ment which must be continued for a year in view of the seriousness of the disorder.

1. Pyloric obstruction is relieved to a large extent in most cases during the first two or three weeks.
2. The pain of the ulcer is completely controlled. Generally it disappears on the first day; seldom it continues for longer than two or three days.
3. Excessive nightly secretion is controlled.
4. Hæmorrhage ceases.
5. Perforation has not been known to occur after the second day during treatment while the patient is in bed or subsequently, if treatment be thoroughly carried out.
6. The penetrating type of ulcer heals rapidly.
7. Ulcers of the stomach and duodenum that have failed to heal after gastro-enterostomy (failure of ulcers to heal after surgical interference is much more common than is generally supposed), are relieved at once of symptoms and eventually healing occurred.
8. The healing of ulcers occurs. Autopsies in patients who have died of pneumonia or other intercurrent disease while under treatment, have shown that ulcers had healed or were in process of healing.

It seems to me that Sippy's plan of treatment is based on sound principles. His experience has been large and his observations have been carefully checked by the results of X-ray examination and analysis of the stomach contents during the course of treatment. Hitherto the medical treatment has not always been based upon an accurate diagnosis and its results have not been confirmed by accurate X-ray examination. These observations cannot be levelled against the method recommended by Sippy. I, therefore, urge you to give this mode of treatment a fair and honest trial.

Addendum.

In the preparation of this paper free use has been made of the articles by:

Hurst: *The British Medical Journal*, April 24, 1920.
Moynihan: *The British Medical Journal*, December 13, 1919.
Moynihan: "Duodenal Ulcer," Second Edition, 1912.
Bolton: "Ulcer of Stomach," 1913.
Sippy: "Disease of Stomach: Practical Treatment," by Musser and Kelly.
Sippy: "Gastric and Duodenal Ulcer," *The Journal of the American Medical Association*, May 15, 1915.
Sippy: "Gastric and Duodenal Ulcer," *Oxford Medicine*.

SOME ASPECTS OF VASCULAR HYPERTONUS.[1]

BY HAROLD J. RITCHIE, M.B., CH.M.,
Honorary Assistant Physician, Sydney Hospital.

WHEN your President did me the honour of suggesting that I should read you a paper I accepted the invitation with mingled feelings, inasmuch as I realized that the request carried with it an obligation which a man in active practice is loath to assume at short notice. I was asked to suggest a subject at once, as the ever-exigent printer was clamouring for his copy, so, rather on the spur of the moment, I suggested that possibly the members of this Association might care to discuss come of the aspects of vascular hypertension. I propose, then, with your permission, to outline some of the commoner varieties of this condition, to discuss briefly its pathology and to consider more thoroughly the question of treatment.

[1] Read at a meeting of the Western Medical Association (New South Wales) on October 20, 1921.

Hypertension and Arterio-Sclerosis.

To many men hypertension spells arterio-sclerosis, but I would remind you that, just as vascular hypertension may exist for some time without producing arterio-sclerosis, so a certain type of arterio-sclerosis may accompany a man to ripe old age without any complicating rise of blood pressure. This latter variety, which embraces probably 50% of the cases included under the general heading of arterio-sclerosis, is generally seen in its most characteristic form in the rigid and calcifying peripheral vessels of old age. The brunt of disease in these cases has fallen upon the larger arteries, the smaller ramifications of the vascular tree suffer less and in many cases are but slightly affected, whilst the arteriolar mechanism is still functioning with more or less accuracy. Examples of a ripe old age with normal mental and physical faculties, though exhibiting extensive changes in the larger peripheral vessels, must occur to all of you. Not to them come the catastrophic accidents in the cerebral vascular field, which so often terminate the usefulness, if not lives, of some of those eminent in every domain of social effort. Rather do their days end through the intervention of an infectious disorder, through the gradual malnutrition of the heart or they may, after passing through "the slow gradations of decay," die from a cerebral thrombosis or rarely from a cerebral hæmorrhage. In such cases systolic pressures rarely exceed 160 mm. Hg. and in many instances never register so high a figure.

The type of arterio-sclerosis consequent upon high blood pressures presents a different picture. It is often a disease of the middle decades of life and unfortunately its onset and course are so insidious that not infrequently irreparable damage has been done ere its presence is disclosed by some chance, such as a life assurance examination.

Persistent high blood pressures are encountered in several conditions, notably in chronic interstitial nephritis. The high pressures of this disease I do not propose to consider, except for the purpose of pointing out that the presence of albumin in the urine, when associated with an hypertrophied heart and raised systolic pressures, is not sufficient for a diagnosis of chronic interstitial nephritis. As a matter of fact, such a diagnosis is not justified in the absence of definite signs of renal inadequacy, such as an excess of urine of low specific gravity, an alteration in the time of the urinary tide as signified by nocturnal frequency and the continued presence of granular casts in the urine. Accompanying these signs we should expect to find some degree of anæmia and sooner or later clinical indications of non-protein nitrogen retention and of some interference with the excretion of sodium chloride; in other words, signs of uræmia.

Now there is a large group of cases associated with persistent high blood pressures which never

presents any indicia of severe involvement of the kidney; albumin may or may not be present, but the specific gravity of the urine is normal or high and granular casts are rare or absent; nor may these patients at any time during the course of their disease present symptoms referable to deficient renal activity. In many cases they are florid, often of a stout habit and belong as a rule to the class who in their own language "do themselves well." But this is not invariable, as at times the ascetic or the elderly spinster, whose lives have been encompassed within the maxim of "plain living and high thinking," suffer from the physical penalties of excessive pressures.

These cases I propose to class under the heading of essential hypertension and to this particular morbid process I wish to call special attention. It is often confounded with other forms of arterio-sclerosis, such as the involutionary or senile type, in which high pressures are absent, and it is frequently dubbed chronic Bright's disease. From the involutionary type of arterio-sclerosis it differs in site, character, extent, progress and prognosis; from chronic interstitial nephritis it differs largely in prognosis, symptomatology and mode of termination. But whilst drawing these distinctions between involutionary arterio-sclerosis and essential hypertension and between essential hypertension and chronic Bright's disease, I would add that there are many cases in which involutionary disease becomes engrafted on hypertensive arterio-sclerosis and many cases of the latter in which the terminal stages present more or less unequivocal indications of superadded chronic renal disorder, whilst in most if not in all long-standing cases arterio-sclerotic changes occur in the kidneys, though the characteristic symptoms of chronic interstitial nephritis are absent.

In its earliest stages essential hypertension is not associated with any structural vascular change, but as the disease progresses, a diffuse arterio-sclerosis of the smaller arteries and especially of the arterioles makes its appearance. The high pressures become inveterate, the larger vessels eventually become affected and the heart is found to be hypertrophied.

Ætiological Considerations.

The original causes of the disorder are subtle and often far to seek. Age, heredity, over-eating, nervous strain, pressor toxins elaborated by metabolic error, the infections, imbalance of internal secretions have all been cited as playing varying parts in its production. It is probable that changes in the capillary bed, "where the business of life takes place," are the original source of the increased blood pressure. Obscure processes, both physico-chemical and bio-chemical in nature, convert what was originally a sandy loam capable of rapid and efficient irrigation into soil of a clay-like consistence whose clogged pores demand high pressures for efficient nourishment. Now the capillary bed depends for its varying needs upon the arteriolar supply and an increased demand is promptly met by an increase in pressure.

The various factors in the production of this increased call of the tissues may be briefly considered. Age, as might reasonably be expected, plays a part; heredity too—inherited predispositions are a commonplace of medicine. The pleasures of the table specially seem to bring its votaries under the ban of high pressures. The exact mechanism by which over-indulgence in nitrogenous food brings this about is by no means certain, but it can be stated with confidence that the majority of sufferers will admit that at least they have been hearty eaters. Gluttony has proverbially found its termination in an apoplexy. "There is death in the pot!" Possibly a definite pressor toxin is evolved from the incomplete metabolism of the proteid molecule, though most of such products seem to be rather depressor in action. Recently, however, Barger and Dale have evolved from this source a highly pressor substance only inferior to adrenalin in potency.

The rôle played by alcohol might be described in the legal phrase as "accessory before and during the act." Over-indulgence in alcohol alone does not seem to create high pressures; indeed, the tension in alcoholic cirrhosis of the liver is, in my experience, moderate or low. Persistent nervous strain is frequently one of the precursors of hypertension, nor can it reasonably be denied that a vicious stimulation of the vaso-constrictor mechanism from psychical causes may play a considerable part in the production and perpetuation of high pressures. The infections, more particularly those of a chronic nature, such as syphilis, whilst causing ravages of the most serious character in the cardio-vascular mechanism, do not apparently predispose to hypertension. Nevertheless, the coincident presence of syphilitic arterial change makes the prognosis in essential hypertension much more serious. Chronic plumbism causes chronic interstitial nephritis rather than essential hypertension.

Much has been written of the action of the internal secretions in the production of this disease, but no definite proof has, as far as I know, been brought forward to substantiate the claims that there is adrenal hypertrophy or that the pituitary or thyreoid secretions are in any way to blame.

The only type of hypertension in which endocrine imbalance has been proved to play a part, is that met with in women about the climacteric, which often yields rapidly to the administration of extract of *corpus luteum*.

I do not feel competent to discuss the question of increased viscosity of the blood, as it involves physico-chemical problems which more properly belong to the domain of the bio-chemist than the clinician, as does also the investigation of the part played by plethora in production of increased tension, except to say that within the last year or two I have seen two patients with polycythæmia in whom the red cell count was well over 9,000,000 per cubic millimetre and in whom the pressures ranged from 180 to 210 mm. Hg.. In these cases the enormous increase in the number of the red cells must have caused through increased viscosity alone a considerable rise in pressures, which is of interest in that the class of patient we are discussing, unlike the chronic nephritis, also frequently shows some degree of increase in the number of red cells.

Symptomatology.

Once hypertension from any cause has become established, structural vascular changes are not slow to follow. They arise chiefly in two ways. Hypertrophy of the muscular coat is the primary change; then follows the results due to the mechanical insult offered to the walls of the vessels by the increased pressure. The stresses and strains applied to the intima are such as to interfere with the elasticity and resilience of the intima with sub-intimal hyperplasia and late molecular and cellular degenerative changes. The second factor is that hypertension not only implies raised systolic pressures, but, what is more important, raised diastolic pressures in addition. Inasmuch as the nutrition of the vessels depends on the diastolic filling of the *vasa vasorum* during the elastic recoil of the vessels, the imperfect blood supply afforded by the cramped *vasa vasorum* brings about fibrotic changes in the hypertrophied media. In this manner a diffuse arterio-sclerosis of the smaller vessels is brought to pass. Nextly, the larger vessels and the heart pay toll and the heart enlarges to the left. The requisite conditions for the production of the clinical phenomena are now present. These assume two chief forms: cardiac or cerebral.

The cardiac symptoms of hypertension are those of fatigue and failure, often slight at first. In such cases the heart, having fought the good fight, reluctantly yields to a tide of rising pressures. Possibly some sub-sternal discomfort on exertion, a distressing sequence of extra systoles or the presence of a minor degree of œdema first brings the patient to his physician. If neglected, these symptoms are but the overtures to serious defeat of the heart. Occasionally the first warning recognized is an attack of nocturnal dyspnœa, the so-called cardiac asthma; in some instances these attacks are insistent and repeated.

The cerebral symptoms may take the form of headache, vertigo or tinnitus. On the other hand, a fatal cerebral hæmorrhage may be the first indication of disease. This, I believe, is rare. Not infrequently a series of minor apoplexies, indicated, perchance, by a temporary monoparesis or even an aphasia measured in no more than moments, bring the patient to us with his heart in his mouth for confirmation of his fears. Long before these symptoms appear a neurasthenia may suggest to his medical adviser the necessity of measuring the blood pressures and their range, for not seldom an otherwise inexplicable nerve-fag finds its origin in such a cause.

Vascular catastrophes occur almost always in ill-supported vessels. Happy is he whose nasal vessels yield first. A salutary epistaxis is at once a danger signal and a relief to the over-burdened circulation. Very occasionally an intestinal hæmorrhage occurs. One of the great political figures of Federation days, whom I saw several times in consultation, died after a second large hæmorrhage from the bowel. His systolic pressure had ranged in the neighbourhood of 200 mm. Hg., but examination of his vessels suggested the presence of an accompanying involutionary change.

Hæmoptysis, except in small amounts, is a rarity; but some years ago I saw a patient with Dr. H. M. Moran, who had had a serious hæmorrhage from an old tuberculous cavity in association with high pressure, hypertrophied heart and all the outward and visible signs of essential hypertension. In this case I believe an aneurysmal dilatation in one of the vessels stretched across the cavity, yielded to the strain.

The diagnosis of essential hypertension is often incidental to examination for other causes. An hypertrophy of the heart, an accentuated aortic second sound or an unduly sustained pulse-wave may suggest the necessity for further investigation of the cardio-vascular mechanism.

If, after the exclusion of exaltation of pressures due to nervousness, the sphygmomanometer yields a systolic record varying from 160 mm. Hg. to 200 mm. Hg. or over, just suspicion is aroused. When such pressures are constant and the characteristic urinary changes of chronic interstitial nephritis are absent, a definite diagnosis can be made. The tendency towards a moderate increase in pressures with advancing years at times may leave us in doubt as to whether pressures of 160 to 170 mm. Hg. in the fifth and sixth decades of life should be considered as mild grades of essential hypertension. The fact that the pressures in such cases tend to remain stationary over considerable periods, should not blind us to the realization that the degenerating arteries of old age do not readily withstand any increase in burden.

In hospital practice it is not uncommon to encounter essential hypertension for the first time when the heart is slowly yielding to the strain. Here the clinical picture discloses the phenomena of a dilating heart *plus* systolic pressures varying from 160 to 200 mm. Hg.. A mitral or a tricuspid murmur is present and the systolic and diastolic pressures are approximating to each other. Before the heart started to dilate, the systolic pressure probably ranged in the neighbourhood of 200 to 230 mm. Hg.. Such conditions are eventually encountered in all cases which do not terminate in a cerebral hæmorrhage or secondary renal disease.

Prognosis.

The prognosis in essential hypertension depends almost entirely upon the stage at which it comes under treatment. Once the tissues have become accustomed to high pressure and diffuse vascular change has occurred, treatment can only be palliative, but in the earlier stages a more hopeful outlook is possible. The keynote to prognosis is the condition of the vessels.

Healthy arteries can withstand high pressures for considerable periods. A proper estimation of the extent of the damage done to the smaller vessels is at times very difficult and depends upon several factors. The symptoms are often such as to indicate cerebral vascular trouble or physical examination may reveal an hypertrophied heart and changes in the larger vessels which justify us in the assumption that severe injury has been inflicted on the arteriolar mechanism. The most unequivocal indication, in my opinion, is a raised and rising diastolic

pressure. The normal diastolic pressure is in the neighbourhood of 90 mm. Hg.. This represents the continuous strain which the normal radial vessel has to bear. In essential hypertension, however, diastolic pressures range from 100 to 130 mm. Hg. or even higher. Such pressures, if long continued, spell irrevocable injury to the vascular tissue and presage cerebral hæmorrhage or heart failure.

Treatment.

Treatment varies in the different stages of the morbid process, but in all stages it is essential that the diet should be restricted and elimination encouraged. For this reason the prognosis tends to be slightly better in the obvious gross feeder if he can be persuaded to limit his diet than the meagre eater, who nevertheless also benefits by dietetic restriction, though not to the same degree. Whilst it is the part of wisdom to diminish the protein intake, it is well not to fall into the error of substituting a carbo-hydrate excess in its stead. Not a few of these patients tend to develope glycosuria if care be not exercised in this regard. The general principle governing the dietary should consist in a gradual limitation of all kinds of food rather than the partial prohibition of any one variety. I am afraid that my confession of faith would not allow me to admit that red meats are fraught with any more danger to the patient than white meats, except in so far as their more savoury nature tends to encourage appetite. The purins, however, seem still to preserve their proud status as alimentary pro-toxins and if the medical attendant considers it desirable, a satisfactory diet may be prescribed from the purin-free foods. The golden rule, however, is to insist upon dietetic parsimony whatever foods be eaten. Alcohol should, if possible, be completely prohibited.

If the patient be a man of affairs and burdened by worries, a prolonged holiday under suitable surroundings is often of great and enduring value in the earlier stages of the malady. In the later stages it is essential that mental and physical over-strain should be avoided and it will often test the ingenuity of the doctor to persuade the patient of the necessity for great diminution of his physical and mental activities without exciting a hypochondria which may convert an active individual of manifold interests into an introspective invalid. Here a word of caution may be inserted as to the danger of too-frequent pressure readings, as the patient is apt to be unduly depressed by pressure fluctuations which are of no prognostic moment. Therefore, be chary of talking pressure figures at all to the invalid and, generally speaking, adopt as cheerful a view of his condition as can possibly be justified by physical examination.

Moderate exercise is often beneficial, but any form of effort which is likely to throw a sudden strain upon the heart, is to be deprecated.

Drug treatment is primarily eliminative. These patients do surprisingly well on some form of mercury. The blue pill and black draught of our ancestors, by a timely depletion, have saved many a patient's lenticulo-striate vessels from rupture. These should be repeated from time to time and the *primæ viæ* kept open in the interim by some mild laxative. Or, if it is wished, 0.03 to 0.06 gramme of calomel may be given nightly for a week and repeated at intervals of a fortnight or three weeks. One of the fashionable varieties of saline may be needed each morning. Such treatment, coupled with dietetic restriction, often works like a charm, whilst sweat baths, though not unattended with risk, sometimes seem useful adjuncts to the treatment.

Potassium iodide exhibits its virtues most satisfactorily in those cases in which a syphilitic taint is present. Though I frequently prescribe it, I must admit that I have seen very little benefit follow its use in other cases. The whole gamut of vaso-dilator drugs has proved disappointing in my hands. *Liquor trinitrini* possesses a definite virtue in the breathless attacks of the later stages of the malady and when combined with small doses of bromide, will serve to eliminate the spasmodic element in the hypertension of the nervous individual, but its action is evanescent. The same verdict holds good with regard to the nitrites. It should be recognized, moreover, that the action of the vaso-dilators is purely symptomatic, that at best they but tide the patient over a crisis and that, at worst, once high pressures have become inveterate, if they act at all, they may, by lowering the pressure suddenly and unduly, rob the tissues of the blood supply vicious custom has taught them to need. Nor are they the best drugs at our disposal for the dyspnœic crises of the disease. At such times morphine is our sheet anchor. Not only does it bring a great measure of physical and mental relief, but it also reduces the pressures for a very much longer period. Bloodletting is not seldom a very satisfactory means of lowering pressures and relieving symptoms. It is of more service in plethoric, florid subjects than in the meagre type of patient and is often successful in relieving the premonitory cerebral symptoms—headache, tinnitus and vertigo—for considerable periods. On several occasions I have removed 450 to 600 cubic centimetres of blood with immediate relief to a congested cerebral circulation. Not infrequently the patient returns, asking to be bled once more, on the first recurrence of symptoms and I incline to the belief that I have neglected to perform venesection in quite a number of instances where it would have proved beneficial. In hospital practice it is not uncommon to encounter these cases under the disguise of a dilated and failing heart, though the pressures still range far above normal. When the patient is in such extremity the two most valuable drugs are digitalis and morphine. It seems paradoxical to prescribe digitalis when the systolic pressures may register 180 to 200 mm. Hg., but combined with rest and purgation digitalis restores the tone of the flagging cardiac muscle and the systolic pressure may actually rise with benefit to the patient. At all events experience has proved that by the use of digitalis we may save these patients from the Scylla of heart failure, whilst still avoiding the Charybdis of cerebral hæmorrhage.

In conclusion, I would say that no one is more conscious than I of the manifold imperfections of this short survey of a very difficult subject and if I have given you "very little bread and an intolerable

deal of sack," if, in other words, I have laboured the pathological aspects of the malady to the disadvantage of the clinical, I have done so in the belief that the basic principle of a rational therapeutics is a study of the morbid processes we hope to combat.

I thank you for the patience with which you have listened to this address, as well as for the honour you did me in asking me to read it.

THE PREVENTION OF TUBERCULOSIS.[1]

BY F. J. DRAKE, M.A., M.B., CH.B. (MELB.),
Mitcham, Victoria.

BEFORE we consider the practicable measures for the prevention of pulmonary tuberculosis, it is necessary to enumerate briefly the means available:

(*i.*) Dispensaries.
(*ii.*) Sanatoria.
(*iii.*) Colonies.
(*iv.*) Homes for patients in an advanced stage.
(*v.*) Boarding houses (hostels) for patients with arrested or partially arrested disease.

Sentiment hitherto has entered too largely into the whole question of treating tuberculosis and too little from the economic side. When one considers that many hitherto quite healthy people contract this disease, resulting in an immense loss to the State, there is no need to emphasize the suffering and economic loss. Our great aim should be the destruction of the tubercle bacillus.

The best means to combat this infection are:

(*i.*) Partial isolation or even complete isolation of all patients in an advanced stage and in a helpless condition.
(*ii.*) Improvement in housing.
(*iii.*) Education of the public how best to avoid contagion.
(*iv.*) Information of the poor patients concerning the best means available for treatment.
(*v.*) Training of doctors in the use of all available means for early diagnosis.

For the prevention of the disease we should find the plague spots and clear them out. Each new centre adds to the ever-enlarging circle for disseminating the infection. The dispensary should accomplish this. The patients with advanced disease should be followed up and the "contacts" examined, to detect the early manifestations of tuberculosis. These may be advised as to the proper mode of living. The conditions under which they live, inducing a lowered resistance to tuberculosis, are as important in regard to the activity of the disease as the actual infection. Preventive measures, such as good ventilation, plentiful food, hygienic conditions of work, acting perhaps indirectly, but slowly and certainly, will tend to abolish not only tuberculosis, but all other infectious disease.

Sanatoria.

These should be established for patients in the early and also in the late stages; but the two should be kept separate. Sanatoria are essentially institutions for the education of the patient and the arrest of the disease consequently; they are applicable to both classes. The sanatorium is only one link in the chain, but the most important link. Patients with advanced disease may be drawn from those domestically or industrially so situated that they are a menace to their associates. A beginning may be made with those in the advanced stages and the accommodation gradually increased. The châlet system commends itself as the most suitable, as it is less expensive than other systems and insures better isolation. The patients with advanced disease should, of course, stay much longer under institutional treatment. Ample grounds are required. Institutions of this character would cope with the disease at its very source. Buildings should be light in structure and inexpensive.

The experiment at any rate may be tried, as the land would not run away and the buildings could be used to add to already existing sanatoria if the experiment failed. The period of stay in sanatoria has been much discussed. Bardswell, of the King Edward VII. Sanatorium, says: "More than one writer has touched upon the relative inefficiency of the sanatorium as a means of curing consumption among the working classes. Experience has shown that three months' treatment in a sanatorium is not sufficient in most cases to permanently arrest pulmonary tuberculosis and that too often the consumptive worker is for a time greatly benefited, only to relapse upon his return to his ordinary conditions of life." His stay should, if possible, be prolonged to six months if the best results are to be attained and in some cases longer.

Compulsion.

If the surroundings of the patient are those highly conducive to continued activity of the disease, he should be segregated by compulsion. Such compulsion means benefit to everybody, including the patients. They cannot be treated efficiently in their own homes, as there is no certainty that our efforts are not wasted and that the directions of the doctor are carried out faithfully. These, as well as the patients in the early stages, require scientific, constant, strict and kindly control. Even in the most advanced stage there is hope of arresting or even completely curing the disease. At any rate, a proportion of these patients will attain a measure of good health and usefulness, provided they recognize their limitations.

Homes for Patients with Advanced Tuberculosis.

These homes should be provided in localities where they are easily accessible to the relatives of the patients. If the patients show signs of improvement, they may be passed on to the sanatorium for patients with early and moderately early disease.

The Colony.

The question of the convalescents in colonies has recently been much discussed. There is a general consensus of opinion favourable to the idea and it is now on its trial.

Varrier Jones writes: "Until someone has the courage, whether by means of the colony or other-

[1] Read at a meeting of the Victorian Branch of the British Medical Association and of the Section of Preventive Medicine on September 7, 1921 (see THE MEDICAL JOURNAL OF AUSTRALIA, January 14, 1921, page 51).

wise, to tackle the mass of infection, that is, the middle cases of men, who can do 30% to 50% of the work of the normal man, it is not much good talking about the eradication of tuberculosis." Preference should be given to those intending to take up an open-air life. The after-care of these is really an industrial problem and it is a matter for consideration of the employer, employee and the benefit societies. Many with continued care after leaving the colony with the disease partially arrested would become cured, that is, the disease becomes permanently arrested.

The general routine of the colony should be on the lines of sanatorium life, namely, fresh air, good food and plenty of it, work according to the capacity and ability of the inmate, regular hours and strict medical control.

Good land is essential, markets easily accessible, convenient and quick means of transport. Remuneration according to the value of the work should be given. The fact that the patient realizes that his new conditions are favourable for his good health preserves his self-respect by his ability to contribute something to his support and is not dependent altogether on the charity of others. Return to the colony should be allowed.

Among the occupations suitable for this class may be mentioned: market gardening, poultry farming, forestry, nursery gardening, cultivation of flowers, herbaceous plants, joinery, cabinet making, basket making, bookbinding, boot making and mending, dressmaking and accountancy, etc.. Many of these patients, after leaving the colony, may be induced to live with their families in close proximity to country towns, where the members of their families can find suitable occupation.

Small allotments, say, of two or three acres, may be obtained for them where the bread-winner is the sufferer.

Difficulties of the Colony.

The difficulty in inducing patients to go to the colony are:

(*i.*) Persons in the early stages of consumption are loath to change their occupation.

(*ii.*) Limited capacity to take up new work. This may be partially obviated by giving work as nearly as possible like the previous occupation, but, of course, under suitable conditions.

(*iii.*) Provision for the dependants. This is often a serious factor.

The labour bureau and friendly societies may be used to find suitable work in suitable surroundings.

The Tuberculous Mother.

Pregnant women should be prohibited from working three months before and for three months after confinement. The ladies' benevolent societies could be utilized in making provision for these cases. Means should be available for teaching the economic value of foods and the best methods of preparing them.

Hostels.

The establishment of hostels is a matter in which I am particularly interested. Many of my patients, after leaving the sanatorium with the disease arrested or greatly improved, wish and are able to return to business, yet at the same time cannot live altogether a town life. They desire to spend their leisure hours in a manner as nearly like the sanatorium mode of life as possible. This is necessary for the permanent arrest of the disease. They cannot obtain suitable conditions in the ordinary boarding-house nor in their own homes. If they go to a boarding-house in a suitable locality the treatment must be carried on surreptitiously and therefore inefficiently. Therefore, they become a danger to their companions, passing from boarding-house to boarding-house, leaving a trail of infection behind them. They inevitably become careless in their habits, which is detrimental to their own health and a menace to that of others. I know of many capable nurses with the necessary sanatorium experience who are willing to establish homes for persons of this class (especially unmarried business men and women) and who would be able to carry out the hygienic life so necessary for the maintenance of their health. These homes should be available in each of the outer suburbs. The Board of Health should be approached to license these places and carry out the necessary supervision.

Specialized Training of Medical Men.

Dr. Batty King, of the Royal Chest Hospital, London, states: "What is required is that all available resources for giving intensive and yet comprehensive practical instruction in tuberculosis should be organized and placed within the reach of all who wish to specialize in the subject and any others who would care to avail themselves of the opportunity." Post-graduate classes should be instituted for the early diagnosis of tuberculosis and be followed by clinical instruction at the public sanatoria.

I also propose that a scholarship should be founded and awarded for the best paper during studentship on the prevention of tuberculosis. I know of a gentleman willing to start the fund with a contribution of one hundred guineas. A thousand pounds should be sufficient for this purpose.

I am glad to have this opportunity of bringing before the Victorian Branch of the British Medical Association these few suggestions for the fight against tuberculosis and hope they will be helpful in the cause which we all have so much to heart.

Reports of Cases.

MALIGNANT OVARIAN CYST WITH SPLENIC, PERITONEAL AND PLEURAL METASTASES.[1]

BY ALFRED AUSTIN LENDON, M.D. (LOND.),
Lecturer on Obstetrics, University of Adelaide.

History.

MRS. P., *ætatis* 35 years, first came to me on March 1, 1904, on account of a hypogastric swelling. She had been a school teacher before her marriage on December 16, 1903. As a young woman she suffered from painful diarrhœa and from painful menstruation. The diarrhœa had lat-

[1] Read at a meeting of the South Australian Branch of the British Medical Association on November 24, 1921.

terly been replaced by constipation which required laxatives. With regard to the menses, which were regular as to time, she had always lost a good deal and had usually passed clots. From the first the pain had been severe, but not sufficiently so to cause her to lie up. At the age of twenty-five years she had consulted a roving Yankee semi-philanthropic-semi-educational medical lecturess, who charged her nothing for advice, but merely five guineas for a spring pessary worth, say, 7s. 6d.. When having her trousseau made some two months before her marriage, she sometimes found her self so swollen in the lower part of the abdomen with wind as she supposed, that the fitting-on could not be proceeded with.

On February 28, just ten weeks after her marriage, she was going to a friend's wedding, when she found that her own wedding dress was much too tight; the abdominal enlargement was now also evident to onlookers. On examination next day I found no indications of pregnancy, but a tumour reaching nearly to the umbilicus. The patient was tall and spare, weighing only 53.5 kilograms. A noticeable feature was her somewhat muddy or bronzed complexion.

First Operation.

On March 7, 1904, ovariotomy was performed. When the peritoneum was incised no serous fluid escaped, as so usually happens with ordinary ovarian cysts, but some solid jelly of golden hue. As it was possible that an adhesion might have been mistaken for the peritoneum and a thin-walled cyst punctured, the opening was enlarged cautiously and the ruptured cyst found surrounded by much of this "colloid" material. The hand was introduced to see if there were any adhesions, but on an attempt being made to lift the tumour, it suddenly gave way; handfuls of the jelly escaped from its interior and were ladled with the hand out of the abdominal cavity. Finally the collapsed remains of the torn cyst were delivered, which was found to spring from the left ovary. The pedicle was ligatured and the abdomen well washed out and then closed. A glass tube was left in the lower end of the wound for seven hours and then replaced by a smaller rubber tube, which was removed next morning. A posterior colpotomy was also performed to insure perfect drainage.

The patient left for her home in three weeks' time, the immediate recovery from the operation being quite satisfactory; but at the end of three months her condition was such as to give me anxiety. She suffered from severe pains in the abdomen and the spine. Visions of malignancy floated through my mind and of infection of the peritoneal surface with the "colloid" material, if that were possible. The patient was confined to bed; nothing seemed to relieve the pain, whether applied externally or administered internally or injected subcutaneously. Nothing, however, could be felt in the abdomen. In despair, I applied to my friend, Mr. Dobbie, for his help and he, being unable to undertake the case, recommended Mr. Tredrea as a hypnotist. In a fortnight's time, without ever having put her actually to sleep, Mr. Tredrea relieved her of her pain. Six months after operation she was very well, the periods being natural and painless. In December her weight was 51.7 kilograms.

Second Operation.

Two years later she became conscious of a return of the abdominal swelling, attended with considerable pain and a difficulty in getting about. On examination there was found a tumour as large as the previous one.

A second operation was performed on November 16, 1906. As I was leaving Adelaide for a short holiday, I asked Dr. J. C. Verco to operate for me. The conditions were very much as on the previous occasion and the "colloid" matter was, as before, spilt all over the abdomen. The recovery was rapid and this time there was no need for the hypnotist's services.

For four years she remained well, except for some rheumatism of the knees in 1910, but at the end of this time she began to have attacks of "cruel pain," which she thought were due to pleurisy. In July, 1911, she noticed a fullness in the left upper segment of the abdomen, but again deluded herself with the idea that it was "wind." I saw her on September 28 and found a considerable tumour, with evidence of local peritonitis. It corresponded with a splenic enlargement and the diagnosis seemed to rest between a malignant mass and an abscess, possibly a suppurating hydatid. With a few days' rest in the private hospital the pain disappeared and the tenderness became much less. The cachexia was more noticeable than before.

Third Operation.

The tumour was explored on October 3, 1911, through a large incision through the left rectus. When the peritoneum was opened, a little of the usual "jelly" escaped. The incision was enlarged and a considerable white patch was noticed on the surface of the greatly enlarged spleen. There was abundant evidence of peri-splenitis and parietal peritonitis, which explained the attacks of acute pain. On introducing the whole hand no recurrent growth could be felt in the pelvis. On further examination the spleen appeared to be universally, though not densely, adherent to the diaphragm and back of the abdomen. It was elastic and semi-fluctuant to the touch. The question of how best to remove the organ was the difficulty, as the abdominal incision could not be increased with any advantage. Thinking that there might be some fluid contents, I incised through the white patch. To my surprise there was no bleeding, but much "colloid" stuff escaped. Then, with my hand behind the spleen, I squeezed out a vast amount, whilst gently separating adhesions. Finally I managed to deliver the spleen through the wound, but not before I had perforated it with a finger, giving rise thereby to a little hæmorrhage. The pedicle was controlled and the spleen removed. The splenic arteries were tied, their size being astonishingly small. Some adhesions to the omentum required attention and ligature. Much loose jelly was mopped out of the subphrenic hollow, which was irrigated. The wound was closed without drainage. There was but slight shock subsequently. In a week the patient professed herself to be quite well and she left for her home in less than a fortnight.

Fourth Operation.

For nearly a year the patient continued to enjoy very fair health and was able to do her household work and, moreover, she lost to a great extent her cachectic appearance. But the abdomen gradually increased in size and she was more than willing to have it again explored. On October 21, 1912, this was carried out. Very little "colloid" material escaped when the peritoneal cavity was opened, but a large quantity of dark-stained serous fluid. A counter opening was made for a drain in the left flank and the central incision closed. No tumour was felt with the finger, but a mass of thickened omentum and matted bowel. She returned home in ten days' time feeling herself to be much relieved.

Subsequent Course.

The relief was not of long duration. The tumour steadily increased in hardness as well as in size, till it seemed to occupy the whole central portion of the abdomen and its nodules could be plainly felt through the thin parietes. The flanks were soon found to be filled with separate collections of fluid. From the right side four and a half litres were removed on March 12 and from the left two and a quarter litres a week later. On April 15 both sides were tapped simultaneously and five and three-quarter litres of fluid were withdrawn. The cachectic bronzing became once more very pronounced; chronic obstruction of the bowels and dyspepsia aggravated her weakness. On April 22 a form of pseudo-angina supervened and was the herald of the end. So little did she, however, appreciate her state that a day or two before her death on April 23, 1913, she discussed some plans about a house.

Discussion.

Those who wish for information with respect to this malignant variety of ovarian cyst, should consult an admirable paper by Dr. Thomas Wilson, of Birmingham, published in the October, 1912, *Reports of the Royal Society of Medicine* under the heading of "Pseudo-Myxoma Peritonei." My personal experience is limited to two instances of the disease. Some twenty years ago I assisted a colleague to operate upon a case similar to mine, where on opening the abdomen the cyst was found to have been ruptured. The operator took great pains to get rid of all the jelly that had escaped from the cyst by flushing out the abdomen with lotion, but the patient died after a few hours, apparently of shock.

When, therefore, I opened the abdomen in this present case, I was at once struck by the resemblance. And when, after the patient's removal home, she began to suffer from severe pain in the back, I imagined that she must have a general peritoneal infection; but this diagnosis was premature by several years.

The cause of the rupture of the cyst in a given case is difficult to determine. The walls of the cyst are so thin that it is conceivable that it may be easily ruptured by the necessary manipulations of bimanual examinations or of preparation for operation or that it may be due to straining such as occurs during defæcation. In favour of a quiet, spontaneous rupture of the cyst wall is the fact that no symptoms appear to attend the actual rupture nor apparently is it ever suspected before operation. It may be that the cell contents of the cyst have the power of eroding their own enclosing walls.

In studying the histories of some fifty-seven cases tabulated by Schumann, of Philadelphia (*Surgery, Gynecology and Obstetrics*, 1908) and the seven cases mentioned by Wilson, I have not come across any case in which mention is made of the invasion of the interior of any solid viscus of the abdomen at all comparable to mine, in which the spleen was converted into a multilocular cyst, precisely like an ovarian cyst, there only remaining a trace of the former splenic parenchyma in the capsule. In two cases the appendix seems to have become cystic and considerably enlarged. In any form of ovarian disease symmetry is not uncommon and, therefore, it is not surprising to find that in eleven cases out of the sixty-four there was similar affection of the opposite ovary, discovered either at the time or later on. The rate is probably much higher, as so many of the patients whose cases have hitherto been reported, have died soon after operation, either from shock, sepsis or toxæmia. In one instance on the abdomen being opened both ovaries were found to be cystic and to have ruptured, but usually there is either a history of a previous ovariotomy without any mention of the nature of the cyst or else there is a tale of a recurrence of pseudo-myxoma in the other ovary, which was healthy in appearance at the time of the first operation. The lesson is obvious that in cases where an ovarian polycystoma has ruptured and poured out its pseudo-mucincus contents, both ovaries should be removed at the first operation. Had it not been for her recent marriage, I should probably have done so and now I regret that I did not do so, as the *débacle* might have been indefinitely postponed.

With regard to prognosis, I am very sceptical as to the results hitherto published. My case would surely have been included as a cure, if published after either the first or second ovariotomy. There was at the time no suggestion of the so-called *pseudo-myxoma peritonei* and it was not till five years later that I removed the spleen. Even then, with a very long abdominal incision, there was no peritoneal infection seen. I should therefore be inclined to propose at least five years' freedom from symptoms as the test of non-recurrence.

The last and most distressing phase of the case need not detain us long, nor the diabolic malignancy and the vicious manner in which the growth throttled the intestines and embedded the liver within the short space of a few months. Some writers indeed have treated this affection as though it were primarily of the peritoneum and hence the name of *pseudo-myxoma peritonei*, which surely ought to be discarded. It is said that a similar affection of the peritoneum is met with subsequent to a primary affection of the appendix, but of this I know nothing. The peritoneum's attitude towards the escaped jelly is seen to be at first one of indifference; the jelly is loose in the cavity, not attached to the peritoneum. Later on there is seen to be some evidence of resentment on the part of the peritoneal surface. The peritoneum becomes thickened and it seems to be attempting to absorb the gelatinous material or to encapsulate it (Wilson). It is possible that it alters the constitution of the jelly so as to allow of its absorption into the lymphatic spaces, but whether it does so or not, the jelly seems often to survive the process of encapsulation; if strands of the living epithelial cells enclosed in the jelly are implanted on the surface, it is easy to understand how a metastasis takes place. Some toxin presumably is absorbed into the system from the original ovarian cystoma, for the cachexia was marked from the very first in my case. The metastasis was not limited to the peritoneal cavity, for we found it in the left pleural cavity. In the case of Professor Taylor's patient, it was also found in the middle lobe of the right lung. But, as I say, undue importance is given to this peritoneal phase. When it supervenes, the outlook is hopeless. All our efforts must be directed to early and thorough eradication of the source of this malignant affection.

References.

McDonald: *Edinburgh Medical Journal*, 1885, Volume XXX., page 1,024.
Lapthorne Smith: *American Journal of Obstetrics*, Volume LIII., July, 1901.
Hurst: *American Journal of Obstetrics*, 1906, page 345.
Bellmann: *American Journal of Medical Sciences*, 1893, page 444.
T. Wilson: *Transactions of the Royal Society of Medicine*, November, 1912.
Schumann: *Surgery, Gynecology and Obstetrics*, 1908.
Atlee: "Diagnosis of Ovarian Tumours," 1873, pages 372-4.
Thornton: *Transactions of the Pathological Society of Great Britain*, 1877, Volume XXVIII., page 189.
Smith: *Medical Times and Gazette*, May, 1878.
Griffith: *Transactions of the Obstetrical Society*, 1886.
Eve: *Transactions of the Pathological Society*, 1886.
Spencer Wells: "Disease of the Ovary," 1872, page 55.
Thornton: *Medical Times and Gazette*, February 19, 1881.
Williams: *Transactions of the Obstetrical Society*, Volume XXIV., 1882, page 93.
Doran: "Tumours of the Ovary," 1884, pages 15 and 109.

Pathological Report.

By C. T. Champion de Crespigny, M.D. (Melb.), M.R.C.P. (Lond.),
Lecturer on Practical Pathology, University of Adelaide.

Macroscopical Examination.

The spleen is almost spherical in shape, about the size of a fœtal head, but rather flattened from left to right, and its notches have disappeared. The capsule is thick and hyaline over the greater part of its surface and there are scanty adhesions on its diaphragmatic surface. The blood vessels have not shared in the enlargement of the organ, but on the contrary are quite small and atrophic. In consistence the spleen is soft and pultaceous.

On section the bulk of the organ is seen to be the seat of morbid change. The true splenic tissue exists for the most part merely as a shell one centimetre to four centimetres in thickness. It is tough and of a pinkish colour. Within this shell the rest of the tissue of the organ is replaced by a massive cystic degeneration. Near the periphery the cysts are more or less clearly marked off from each other by fibrous trabeculæ and are from one millimetre to two or three centimetres in diameter, but towards the centre the trabeculæ are broken down and there is one large cystic cavity. The cystic portion is clearly marked off from the more normal part of the organ. In some parts there is no splenic tissue between the cystic portion and the capsule of the spleen itself and over these areas the capsule is much thickened.

The contents of the cysts are a bluish-grey, sticky, gelatinous material, which gives the chemical reaction for pseudo-mucin.

Microscopical Appearance.

Sections of the edge of the growth were cut and stained in hæmatoxylin and Van Giessen's stain. The splenic tissue itself shows fibrosis especially marked along the course of the vessels. The cysts vary much in size, some being quite microscopic. Usually between each cyst and the splenic parenchyma there is a narrow zone of fibrous tissue. In a few places this layer forms the only wall of the cyst, as the lining cells have degenerated and disappeared, but elsewhere a layer of cells form the true cyst wall. In many of the smaller cysts the cells of this layer resemble columnar epithelium, arranged in a single row, with a well marked basement membrane. In some situations, presumably

owing to the pressure of the cystic contents, the cells are flattened and cuboidal in shape. Scattered through the gelatinous contents many columnar or spindle-shaped cells may be seen, evidently shed by the mucous-membrane-like lining of the cysts. On further search cysts are to be found where the lining membrane is thrown into convolutions and the cells are arranged in several layers. Near these are small clumps of the tumour cells, which appear to be infiltrating the splenic parenchyma. These clumps in some instances are breaking down in the centre, as if in the process of forming tiny new cysts. Sparsely scattered beneath the columnar cells lining the cysts are muti-nuclear giant cells. It is difficult to judge whether they are formed from endothelial cells of the splenic tissue or are due to fusion of the tumour cells.

The whole structure of the tumour closely resembles that of a multilocular ovarian cystadenoma, except that (*a*) the lining epithelium is not as regular in its arrangement, (*b*) there are a few areas which show distinct infiltration by tumour cells and (*c*) there are the scattered giant cells which I have never seen in sections of ovarian cysts.

Report of the Autopsy.

The autopsy was performed on the morning of April 23, 1913, about five hours after death.

External Examination.

The body was that of a middle-aged, very emaciated woman. The abdomen was much enlarged. There were several old healed laparotomy wounds. The abdominal enlargement was felt to be due principally to a hard, massive tumour, which extended from the costo-chondral margin above, down into the pelvis and outwards into either flank. Laterally it extended further to the right than to the left; it was in the lumbar regions only that its margins could be felt as hard, nodular edges. The growth was adherent to or continuous with the abdominal parieties, which were not movable over it. Behind the growth on either side the presence of fluid could be detected. There were several small nodules the size of a marble in the laparatomy scars.

Dissection of the Abdomen.

First of all about 2.8 litres of clear, brownish fluid were removed from the abdominal cavity by means of a stab in each flank and a silver catheter.

A median longitudinal incision from the supra-sternal notch to the pubes was made.

Cutting through the abdominal wall just above the umbilicus, the knife encountered the substance of the mass referred to above, which was found to be adherent and even continuous with the parietes. It was therefore necessary to remove the anterior abdominal wall after reflecting back the skin together with the main tumour mass. This was done. The condition of the abdomen will be described first.

The main tumour mass seemed to have originated in the peritoneum lining the anterior abdominal wall. It had caused enormous thickening and had grown around and become adherent to neighbouring structures without invading them to any marked extent. The growth was irregularly wedge-shaped, the base of the wedge being upwards. Into this base the lower edge of the liver was implanted and the growth had extended further upwards, uniting the hepatic serosa to the parietal peritoneum and converting both into tumour substance, thus forming a layer about five millimetres in thickness. Opposite the lower edge of the liver the growth had an antero-posterior thickness of about fifteen centimetres. The tumour in this part of the abdomen had followed the folds of the peritoneum and within it were found those organs or parts of organs which they enclosed. Thus the liver was enveloped. The gall bladder and the structures in the foramen of Winslow, the pyloric canal of the stomach, the pylorus and first three portions of the duodenum, the head of the pancreas, the transverse and part of the descending colon were all buried in the substance of the growth. The great omentum was merged into the mass also. The gastric lumen was dilated, but all the other hollow viscera appeared as mere slits, except the gall bladder, which was full of green bile. All these points were determined by transverse horizontal sections after the tumour had been hardened. The lower limit of the tumour jutted downwards into the true pelvis behind the bladder. The caecum was adherent to but not enveloped by the growth.

On section the tumour substance looked like a mass of firm, partly-cooked sago, but was quite dense in texture and not at all friable. There were no large cystic loculi like those found in the spleen, but the cysts were all very small, just visible to the naked eye, except in one place next to the liver, where they were somewhat larger. The areas of peritoneum not involved in this large tumour mass were inseminated with firm nodules, closely set and varying in size from two or three millimetres to one centimetre in diameter. They were sown all over the mesentery and the serosa of the small intestine. The uterus was small, virginal in type and buried amongst small cysts which sprang from the peritoneum. The urinary organs were normal.

The Thorax.

The pericardium and heart were normal. The lungs were natural, but each pleural cavity contained about fifty cubic centimetres of clear fluid. Beneath the pulmonary pleura in each side there were two or three small cystic growths identical with those in the peritoneum and about one centimetre in diameter.

The Skin.

The scars were incised and the nodules that had been felt in them were found to be small cystic growths which had sprung from the surfaces of the incision in which they had probably been implanted at the time of the operation.

Microscopical Examination.

The tumour possessed a structure almost identical with that described in the splenic growth. It consisted of many small cystic alveoli. These contained a hyaline material which was proved to be "pseudo-mucus." The alveoli were buried with one or two layers of cells, columnar or quadrilateral in shape. The cells often merged into each other, so that the layers seemed to be composed of syncitium in some instances rather than of individual cells. The growth was not very vascular. The blood vessels were in the scanty stroma between the cysts. There was no degeneration present and the walls of the small cysts showed no such tendency to break down and thus to form larger cavities, as was seen in the spleen. The growth, when in contact with a solid organ like the liver, seemed to advance by direct invasion, absorption and replacement of the hepatic substance rather than by infiltration with detached cells of the growth.

Reviews.

THE THROAT, EAR AND NOSE OF CHILDREN.

A new addition to the Edinburgh Medical Series, a small volume by Dr. Douglas Guthrie, is designed to supply concise, practical information regarding the common diseases of the ear, nose and throat in early life in a form understandable by nurses and social workers.[1]

The anatomy of the parts and their affections, together with simple line diagrams illustrating methods of examination and pathological conditions, are described briefly and the diagnosis and treatment required outlined. Commonly used instruments and their method of employment are depicted and assist in elucidating the text.

We are pleased to see emphasized the statement that measles, usually regarded as a trivial illness, is in reality a more serious and disabling disease in regard to its effects on the ear, than all the notifiable infectious diseases together. A few formulæ are given at the end of this useful little book.

[1] "Diseases of the Ear, Nose and Throat in Childhood," by Douglas Guthrie, M.D., F.R.C.S.; 1921. London: A. & C. Black, Limited; Post 8vo., pp. 88, illustrated by 30 figures. Price: 5s. net.

The Medical Journal of Australia

SATURDAY, MARCH 25, 1922.

The Metric System.

In the year 1916 the Council of the South Australian Branch of the British Medical Association recommended the members to adopt the metric system in prescribing. Sir Joseph (then Dr. J. C.) Verco delivered the message from the Council to a meeting of the Branch and adduced the most convincing arguments in favour of the acceptance of this proposal. In the annual report of the Branch it is recorded that it was unanimously decided by the Branch to recommend all the members to adopt this method. A year later the same subject was discussed by the members of the New South Wales Branch of the British Medical Association on the initiative of Dr. F. Guy Griffith. A proposal to request the Faculty of Medicine of the University of Sydney to teach the metric system to the exclusion of the Imperial to students was disallowed because notice of motion had not been given. A month later the motion was carried without dissent. All those who took part in the discussions, recognized that the only way to achieve the objective would be to avoid the use of the Imperial system altogether. In the annual report of the Victorian Branch of the British Medical Association for the year ended December, 1917, it was announced that the Council had recommended to the Faculty of Medicine of the University of Melbourne that the metric system of weights and measures should be taught to the exclusion of any other system. A sympathetic reply had been received and it was arranged that the change should come into force in 1918.

In June, 1917, The Medical Journal of Australia announced its intention of assisting in this belated reform by the sole use of metric weights and measures and the metric or centrigrade system of thermometry. Very little adverse criticism was offered; a few recorded their approval of this action. About this time the Adelaide Hospital Pharmacopœia appeared without the old-fashioned and clumsy dosage in grains and minims. A little later the Melbourne Hospital Pharmacopœia was issued with the old and new side by side. Still later the Sydney Hospital Pharmacopœia adopted the same expedient as the Melbourne Hospital. The gradual introduction of the metric system into pharmacology started in Great Britain in the year 1867, when the two methods were given in connexion with volumetric analysis in the British Pharmacopœia. The same plan was followed in the 1885 edition. In the 1898 edition the Imperial system was discarded in connexion with the preparation and standardization of drugs, but was preserved for the specification of dosage. In the last edition (1914) the metric system is employed throughout, the Imperial dosage being still given in addition.

In medical practice certain therapeutic preparations are invariably prescribed in metric doses. Sera, vaccines, blood for transfusion and the like are administered in doses measured in cubic centimetres or litres. The laboratory worker has no time to waste with the complications of the Imperial measures and weights. He employs the metric system because unnecessary calculations are thereby avoided and the risk of error is materially reduced. Everybody knows that the bacterial incubator is normally standardized to 37° C. as the optimum temperature for the growth of the majority of bacteria. The division of the thermometer into one hundred degrees, with the freezing point of water at zero and the boiling point of water at one hundred is simple and rational. The Fahrenheit scale, with its 32° for the freezing point, its 212° for the boiling point and the interval of 180° between the freezing and boiling points of water, has nothing to recommend its use.

The arguments in favour of the metric system have been set forth so many times that it would be tedious to repeat them over again in this place. Even the strongest opponents to the system admit that in connexion with weights, measures and thermometry it is simple, easy to use and rational. The one reason against its general adoption by the medical profession in Australia is that it entails some trouble

for those who have used the complicated and irrational Imperial dose and measurements, to go through the transitional phase of translation.

Despite the fact that the metric system has been consistently used in The Medical Journal of Australia for nearly five years, it must be admitted that the majority of medical practitioners in the Commonwealth have made no effort to join in the reform. Complaints have reached The Medical Journal of Australia that the metric system is not understood by the majority of readers. The proposal has been put forward that both the metric and the Imperial dosage should be given in these columns and that the Fahrenheit scale of temperature measurements should be indicated. To assent to this proposition would be to admit that the movement initiated by Sir Joseph Verco and his enlightened followed have proved a failure and that there is no prospect of advance by the present generation of medical practitioners. It is obviously useless to supply the translation when a teacher wishes his pupil to acquire a knowledge of a foreign language. A small concession might perhaps be made by giving the old together with the metric doses occasionally, to remind the reader of the equivalents.

The medical profession is comprised of men and women trained in science. It is inconceivable to us that men and women education in science could have any difficulty in making the simple translations necessary for the purpose of prescribing or recording temperature. A gramme is equivalent to nearly sixten grains; a cubic centimetre is equivalent to nearly sixteen minims; a grain is equivalent to approximately 0.06 cubic centimetre. The average temperature of a human being in health is 37° C.; 38° represents slight fever; 39° represents well developed fever; 40° (equivalent to 104° F.) represents high fever. Surely these equivalents are not difficult to memorize. Should this journal give up its endeavour to lead the medical profession along a sensible path after the Medical Faculties have admitted the advisability of teaching students the metric system to the exclusion of the Imperial and after three important Branches of the British Medical Association in Australia have declared in favour of the reform? Is it not too late to turn back?

DIETING IN PRACTICE.

The average medical practitioner does not devote much time or thought to the food taken by his patient. He ignores the use of scales for weighing the food ingredients and is quite indifferent to the number of calories ingested each day. The question of the provision of an adequate supply of accessory food factors is usually regarded as a theoretical matter, only to be adopted to practical conditions in the presence of definite deficiency diseases. This attitude is unfortunate, since much can be gained from dieting in the treatment of the patient who is suffering from one of the very numerous affections associated with a disturbance of nutrition. A patient losing weight in the course of any disease usually requires much coaxing to induce him to take even a moderate supply of food. The practitioner who is satisfied to order easily digestible and tasty dishes, will rarely achieve an important increase in weight. It has become fashionable to treat diabetes in a patient by starvation according to the teaching of F. W. Allen. Tables of caloric values are used and stereotyped diets are prescribed. It is a relatively easy task to restrict a diet; to build up a diet which will have the effect of stemming a partial starvation, is much more difficult. It need scarcely be pointed out that, even in the case of the diabetic, the patient and not his disease should be dieted. The individuality of the patient must be studied and a careful series of experiments should be undertaken in order that the best results may be attained. In these experiments the assistance of the patient is often of great value. It is unfortunate that tables of caloric food values compiled from analyses of food-stuffs in Australia are not available. Tables constructed in America or Europe have to be used. If the fact be recognized that these tables are not quite accurate for Australian food, this disadvantage will be minimized. In the next place, every article of food eaten should be accurately weighed and the result recorded. A daily entry on the diet chart, setting forth the quantities of proteins, carbohydrates, fats and salts actually ingested and the equivalent caloric values, should be made. The experiment consists in endeavouring to find a diet composed of an appropriate quantitative mixture of

protein, carbo-hydrate and fat which contains nourishment equivalent to a supply of calories in slight excess of the physiological need of the individual at rest in bed or taking mild exercise, as the case may be. It will be remembered that the basic metabolism of a man weighing sixty kilograms is approximately 1,860 calories a day. If pyrexia be present, the supply must be increased. The patient will aid the practitioner if it be explained to him that the heat value of food can be measured in this way and that it is essential to recovery that a certain level shall be attained and maintained.

The importance of making provision for the addition to a diet containing a proper heat value of substances including the accessory food factors will be manifested by experiment. Information concerning the relative value of the different forms of foods in this regard is available in the writings of Gowland Hopkins, Mellanby, MacCollum, Drummond and Zilva, among others. Some very remarkable results can be obtained in the dieting of patients by the judicious additions of small quantities of suitable substances. It is useless to rely on proprietary preparations for the supply of a mixture of so-called vitamins. Since the practitioner is groping for knowledge in any given instance, it is necessary to employ ordinary foods of known value as accessory factors. Dieting on these principles should be carried out by every medical practitioner who recognizes the necessity of treating his patient rather than a disease.

PERIODICITY IN FILARIASIS.

The problem of the ætiology and prevention of filariasis is a matter of serious concern to Australia. Dr. A. Breinl has recently called attention to the fact that filariasis is three times as common among the patients admitted to the Brisbane General Hospital as among those admitted to the Townsville Hospital. The incidence in the Brisbane General Hospital is as high as 15%. Filariasis is responsible for much chronic invalidism and economic loss wherever it occurs. Some observers have endeavoured to prove that the infestation of man with the worm itself is of small moment, because they have been unable to trace any direct effects in the vessels of the host. Low and Manson-Bahr have questioned the justification for this attitude and have claimed that no helminth inhabiting the arterial or lymphatic vessels can be harmless. They have discovered evidence of the local influence of filariæ on the vessel walls and on the lymphatic glands. All observers are agreed that filariasis is ultimately a destructive and crippling disease, even if the complicating elephantiasis be excluded. If this disease is a serious menace to the Queensland people, it is a much more serious menace to the people inhabiting the Pacific islands. The incidence in some of these islands is alarmingly high and its effects are appalling.

In order that measures may be taken to cope with filariasis, more knowledge must be collected concerning its ætiology, pathogenesis and transmission. One of the points which requires special attention is the nature, cause and variations of the periodicity. It is well known that the embryos of *Filaria bancrofti* appear in the majority of tropical countries in the peripheral blood vessels of the human host only at night-time. Dr. J. P. Maxwell has studied this among other aspects of the filariasis question in China and has published the results of his observations.[1] He points out that it is unknown when the embryos appear in the peripheral blood stream after infection has taken place. It is also unknown for how long a period the adult worm is capable of giving birth to embryos. He is of opinion that man may harbour the worm for considerable periods without manifesting any symptoms or signs of disturbance of health. He appears to regard the question of the periodicity as an interesting academic phenomenon. This attitude does not give promise of advance in knowledge of the problems involved in the prophylaxis of the condition. He recognizes two hypotheses as possible explanations of periodicity in filariasis. The first is that there is an accumulation in the body of some chemical substance which, while producing sleep, tends to attract the micro-filariæ to the periphery. Apart from the fact that there is no known chemical substance which could exercise this double function of inducing sleep and attracting minute living organisms to the peripheral blood vessels, this hypothesis must be refused in the absence of any acceptable facts in its support. The second hypothesis is that the capillary circulation is endowed with a mechanism which determines this strange phenomenon. It has been shown that vaso-dilators, such as nitro-glycerine, diminish the number of embryos in the peripheral blood, while vaso-constrictors, like pituitrin, induce an increase in the number of embryos. Allen J. Smith and Damass Rivas put forward the view in 1914 that during the period when the cutaneous vessels are contracted, the embryos appear in large numbers in them as a result of mechanical retention. This view, however, is open to the objection that the distribution of the embryos between the blood vessels in the deep-seated organs and the vessels in peripheral situations would be relative rather than absolute where it is dependent on retention as a result of vaso-constriction and a slowed blood flow. Moreover, there are other circumstances which tend to show that the cause of the periodic appearance in the peripheral circulation is not entirely a quality of the vessels.

[1] *The Philippine Journal of Science*, September, 1921.

Bahr has found that *Filaria bancrofti* in Ceylon, India, the West Indies and many other countries has a periodicity of a definitely nocturnal character. J. E. L. Johnston found in 1914 that *Filaria demarquay* is devoid of all periodicity, while the embryos of *Onchocerca volvulus* appear in the peripheral circulation at irregular times. Bahr recognized that the filaria in the Pacific islands has either no periodicity or manifests this character in an irregular manner. Leiper, working in the north of Nigeria, discovered that the embryos of *Filaria* or *Onchocerca volvulus* did not appear in the peripheral blood stream of persons with tumours due to these worms. He further failed to observe any embryos of this worm in the peripheral blood of monkeys in which he injected living embryos, either subcutaneously or intra-peritoneally. Several competent observers have recorded that *Filaria loa* in Africa appears in the peripheral blood during the day-time and disappears at night. Külz endeavoured to vary the periodicity of filariasis in his own person by various devices. He had been infected in South Kamerun and the embryos of *Filaria loa* appeared regularly between seven and nine o'clock in the morning, reached the maximum numbers at three o'clock in the afternoon and disappeared at nine at night. This regular migration was maintained on his return to Europe and persisted eight years later when he went to New Guinea. He found that the number of embryos in the peripheral blood increased to some extent when he produced a passive hyperæmia of his arm and diminished to some extent when the arm was immersed for a time in cold water. K. Kado and Y. Hara failed to change the periodicity from nocturnal to diurnal by placing the infected individuals under the influence of artificial light at night-time and by requiring them to perform violent exercises at night-time. It has also been noted that, even with infections by *Filaria bancrofti*, the nocturnal periodicity is not universal, at all events in East Africa. Approximately 2% of those infested show a diurnal appearance of the embryos in the peripheral circulation.

It is evident that the assumption of a mechanism inducing a nocturnal periodicity of *Filaria bancrofti* cannot be supported, in view of the diurnal periodicity of *Filaria loa* and the absence of all periodicity of *Filaria demarquay* and some of *Onchocerca volvulus*. In Queensland the worm found is *Filaria bancrofti* and, according to Breinl, the nocturnal periodicity is the rule. The filariasis of the Pacific islands is of a different type. These facts would suggest that the cause of the periodic appearance in the peripheral blood stream is a biological character of the worm. Further evidence is needed before any statement can be made as to why some embryos seek the shelter of the contracted vessels with a sluggish circulation, such as obtains in the peripheral vessels at night during rest, while other embryos prefer to swim in the rapid currents of the peripheral circulation during the period of bodily and muscular activity. At present nothing beyond the mere facts of the periodic appearance of the embryos in the peripheral blood stream is known. The cause of the periodicity is still quite obscure.

SWELLING OF THE ARM AFTER OPERATIONS FOR CANCER OF THE BREAST.

ŒDEMA associated with the blocking of lymphatic channels is a very common sequel to extensive operations in various parts of the body. It is usually held that the swelling is the direct result of the obstruction to the lymph flow and that the greater the disturbance in the lymphatic system, the greater will be the œdema. Dr. W. S. Halsted has allowed a long and large experience to raise doubts concerning the correctness of this view.[1] His attention to the nature of this form of post-operative œdema was attracted many years ago, when the removal of the lymphatic glands from both groins had been followed by a great and permanent swelling of the scrotum. He had seen this patient thirty-eight years after the operation and the scrotum was still large and incommodious. Careful observations of patients who had undergone removal of the breast for cancer, with more or less complete extirpation of the lymphatic glands in the axilla, convinced him that, while obstruction of the lymphatics and of the veins might be to some extent involved in the production of periodic or persistent swelling of the arm, infection was a more important factor. This opinion was confirmed by the facts eilicited in connexion with the history of a patient who had had her breast removed in November, 1916. Three years and three months later, swelling of the arm appeared for the first time. The swelling had been ushered in by nausea, a rigor and high fever, following an attack of influenza. The swelling became distressingly great and manifested all the characters of post-operative œdema. An operation was performed shortly after on account of a small local recurrence involving the second and third parts of the axillary vein. The vein had been occluded before the swelling of the arm had appeared. The vein and the recurrent tumour were removed. Healing took place without visible infection and the swelling disappeared completely.

In 1891 he described the operation now known as the Halsted operation for the removal of the breast. In 1913 he introduced some modifications in the technique, more particularly in regard to the closure of the wound. He found that the swelling of the arm, which had been observed not infrequently after the original operation, became rare after the adoption of the modifications. After the original operation he had noted that extreme abduction of the arm was often prevented by a cicatricial band occupying the situation of the extension of the incision on to the arm. When this defect in the operation was recognized he shortened the incision down the arm by degrees until he found that he could abandon it altogether. The edge of the skin at the upper margin of the wound was fastened with fine silk sutures to the first intercostal muscle and its fascia in such a manner that the axillary fornix was raised to the highest desired point. This alteration in the operation provided him with a super-abundance of skin in the axillary-clavicular region. No

[1] *Bulletin of the Johns Hopkins Hospital*, October, 1921.

attempt was made to approximate the cut edges of the skin in the upper third or half of the denuded area. This area was covered by skin grafts. In this way the band of scar tissue was avoided and the patient was enabled to raise her arm without difficulty. The results of the new technique were excellent. Swelling of the arm sufficient to incommode the patient was no longer encountered. Marginal necroses were rarely seen. The modified operation enabled him to remove the breast and the lymphatic glands and vessels as extensively as before. It was therefore obvious that the absence of swelling could not be due to a diminished obstruction to the lymph flow. He found that by the careful application of Ollier-Thiersch skin grafts, without any tension on the cut edges of the skin, necrosis and infection could be avoided. He not unnaturally concluded that the swelling of the arm after the so-called radical operation for the removal of the breast was primarily the result of infection.

Additional evidence is quoted in confirmation of this doctrine. In the first place, it is now common knowledge that elephantiasis caused by filariasis is not due to simple mechanical blocking of the lymphatic channels. In the next place, Dr. Halsted asked Dr. Reichert to conduct some interesting experiments bearing on this question. All the tissues of the leg of a dog were divided except the femoral artery and vein, the main nerve trunks and the bone. After seven or eight days a slight swelling was noted. As soon as this swelling had subsided, the femoral vein was occluded by ligature. No appreciable increase in the size of the limb occurred. Later the experiments were repeated and a week after the partial amputation, both the femoral artery and the femoral vein were ligatured. Still no considerable swelling was noted. It thus appears that the circulation is sufficiently restored within a week of its arrest by division of all the smaller vessels to prevent the appearance of swelling or gangrene, even after the occlusion of the main artery and vein.

Dr. Halsted is convinced that the attempt to preserve skin for the purpose of carrying out the so-called plastic operations is not only dangerous in increasing the risk of recurrence of the malignant disease, but is prone to be followed by mild or severe infection in the suture lines, despite the utmost care. He has ample evidence to show that the amount of infection may be very small and yet sufficient to lead to swelling of the arm at later periods. Often the attacks are recurrent and are determined by some trauma or other circumstance calling the latent infection into activity. His arguments are compelling and rational. It is inconceivable that, even when all the lymphatic glands are removed from the axilla and infra- and supra-clavicular spaces, the lymphatic circulation is permanently arrested. There is histological evidence that, after the lapse of time, fresh lymph channels appear and it is not improbable that new glands are laid down. If the swelling were due to blockage of the lymph channels, it would be of short duration, whereas every surgeon knows that the swelling appears again and again for years after the removal of the breast. Moreover, mere obstruction of the lymphatics could not account for the coming and going of the swelling—a common phenomenon. It is always worth while to challenge the correctness of well-established dogmas, lest some of them prove to be assumptions devoid of physiological foundation.

CAPILLARY CIRCULATION.

It has long been the dream of pathologists to be able to apply microscopical methods of examination to living tissues. Less phantastic is the aim of physiologists to study the circulation by means of ocular instruments in the healthy and in the diseased. From the earliest times the microscope has been used for observations of semi-transparent tissues of some lower animals. When the ophthalmoscope was first invented by Helmholtz in 1851, it was hoped that information concerning the vascular apparatus would become available. Unfortunately, none of the earlier methods could be applied in such a manner as to illuminate the capillary circulation in the living human subject, so that variations from the normal might be recognized. In 1874 Hueter invented an instrument for the inspection of the vessels of the lower lip. This instrument was still insufficient for the purpose of clinical observation. In 1912 Lombard described an ingenious method of visualizing the capillaries in the cuticle at its junction with the finger nail. A strong, obliquely transmitted light was directed on the junction of the cuticle and the nail and the cuticle was examined by means of a low-power microscope. Weiss and others in Germany have developed this method since the year 1916 and special apparatus have been devised for the purpose. Dr. S. O. Freedlander and Dr. C. H. Lenhart have carried out a relatively large series of observations with this method and, while they are indisposed to draw far reaching conclusions from the observation of the capillary circulation in a very limited area at the terminal portion of a limb, they record some interesting data and invite other workers to confirm or correct their findings.[1] Lombard's method enables the observer to focus a row of capillaries so that the short, narrow, arterial limb, the thick, long, venous limb and the thick, short, connecting limb are plainly visible. They noted that reflex stimuli applied to the other hand caused alterations in the appearance of the arterioles and in the blood flow. In traumatic shock the development of capillary stasis was watched and variations in the capillary loop were studied in all the stages. Similar changes were seen in surgical shock and in septicæmic conditions. Some interesting observations were made in cardiac disease with the clinical signs of decompensation. It appears that stasis is an early manifestation of decompensation. The capillaries visible under the microscope are increased in number, the arterial limb becomes small and narrow and the connecting and venous limbs become wide and full. When further observations on this interesting matter are available, the subject will be discussed in greater detail.

[1] *Archives of Internal Medicine*, January 16, 1922.

Abstracts from Current Medical Literature.

MEDICINE.

Abdominal Migraine.

WILLIAM A. BRAMS (*Journal of American Medical Association*, January 7, 1922) states that, although not mentioned in many works on gastro-enterology, the abdominal type of migraine is not such a rare clinical entity as might be supposed. Only recently has the close relation between certain types of periodic epigastralgia and head migraine received recognition. Some writers have expressed the opinion that the abdominal type of migraine was simply another form in which migraine might manifest itself; others have reported cases in which the epigastralgia was distinctly a temporary and vicarious substitute for the usual form of cephalic migraine. The author has reported observations of twenty-two cases in which all precautions were taken to exclude every kind of organic disease of the nervous system or abdomen. Epigastralgia of this type was distinguished by periodicity, the attacks often having occurred with almost mathematical regularity. The intervals between the attacks were symptomless. There was a history of migraine in either the patient or his family and there was an absence of evidence of gross organic disease. Abdominal migraine may manifest itself in three forms: (*i.*) The ordinary type, in which the epigastralgia is accompanied by head migraine or appears as the only symptom of migraine. (*ii.*) The vicarious type in which typical head migraine has existed up to a certain time, after which abdominal migraine has appeared and remained as the only manifestation of migraine. In certain cases the abdominal manifestations disappeared on the return of the head migraine. In other instances abdominal and head symptoms alternated. (*iii.*) The larval or irregular type, in which the periodic attacks consist of vomiting with epigastralgia less marked. In cases conforming to the first two of these forms, the typical attack begins abruptly, recurs at definite intervals, generally every four to six weeks, lasts for a varying period up to three or four days and ends as abruptly as it began. The interval between the attacks is free from all symptoms. In the first group the attack usually makes its appearance at about eighteen years of age, in the vicarious form usually at the menopause, while no definite age incidence has been noted in the larval types. The large majority of the recorded cases have been in women. The attack usually consists of a severe cutting or boring epigastralgia, generally limited to the epigastrium, but at times radiating in any direction. The vomitus is chiefly of bile and mucus and the pain is not relieved by vomiting. Constipation is commonly present, but diarrhœa is not unusual during the actual attack. The disease is refractory to treatment, but may be favourably influenced by the measures commonly employed in anti-migraine therapy.

Cancer of the Lung.

CREYX (*Journal de Médecine de Bordeaux*, December 10, 1921) points out the difficulty in diagnosis of primary and secondary malignant disease of the lung. He publishes the records of three patients of his own and compares the symptoms and signs with those already mentioned in the literature. His first patient had malignant disease of the stomach. An area of dulness in the chest appeared, followed by hæmorrhagic pleural effusion at the base of the right lung. These signs and the patient's symptoms led to a diagnosis of secondary carcinoma of the lung. At the autopsy a large area of caseous pneumonia was found, with no evidence of neoplasm. The second patient had all the signs and symptoms of tuberculous pleuro-pnuemonia, except that *Bacillus tuberculosis* was not found in the sputum. The autopsy revealed malignant disease of the lung with ulceration. The third case was diagnosed first as bilateral effusion and later as double spleno-pneumonia. The autopsy revealed infiltration of both lungs and pleuræ by sarcomatous masses and extensive gangrene of the left lung. The author points out the lack of a definite clinical picture in published cases and emphasizes the difficulty of diagnosis on this account. The symptoms and signs are extraordinarily variable and though authors lay stress on different signs, they admit that there is no reliable diagnostic sign, except the presence of cancer cells in the sputum, and this is so rarely present that it affords little help. Symptoms, such as hæmoptysis, progressive dyspnœa and cachexia, are common to many affections; signs of consolidation, of mediastinal pressure, of deformity of the thorax, rapid reaccumulation of pleural effusion, bloody effusion and enlargement of superficial lymphatic glands are not diagnostic of cancer. The X-ray picture is variable. There may be a diffuse or a clear-cut shadow; sometimes a pleural effusion obscures the picture; generally the mediastinal glands throw a larger shadow than normal. Contrary to usual experience, fever was present in the author's cases and this sign increased the difficulty of diagnosis.

Leuchæmia and Tuberculosis.

P. EMILE-WEIL AND COSTE (*La Presse Médicale*, November 23, 1921) draw attention to the possibly of *Bacillus tuberculosis* being the infective agent in leuchæmia. They publish records bearing on this hypothesis. The leuchæmias are thought to be due to some unknown infective agent and the question of a tuberculous origin for the leuchæmic syndromes has not been sufficiently investigated. A young man admitted to the Tenon Hospital on December 27 with enlarged cervical glands and hæmorrhage, was profoundly anæmic and each cubic millimetre of his blood contained 54,000 leucocytes, of which 92% were myelocytes. In January the blood count altered, there being 45% lymphocytes and 48% undifferentiated cells; the leucocyte count rose to 90,000. There was constant fever, a double hæmorrhagic pleural effusion and ascites. The man died on March 2. Autopsy revealed tuberculous peritonitis and scattered caseous and miliary tubercles in the abdomen and thorax. No evidences of leuchæmic lesions could be found, except in the liver, the great omentum and the bone marrow, all of which showed lymphocytic infiltration. The enlarged glands were entirely caseous. No giant cells were discoverable at all, but sections showed an acid-fast bacillus morphologically identical with Koch's bacillus. The inoculation of a guinea-pig could not be made. A number of cases have been published resembling the above in the combined affection of the hæmopoietic system by tuberculosis and leuchæmia; another group has been collected by Rietti, in which old tuberculous lesions have been found in patients who died with definite leuchæmia of the lymphoid or myeloid type. In view of this combination, careful investigation should be made in leuchæmic patients to determine the presence or absence of *Bacillus tuberculosis* in the lesions; microscopic diagnosis should be confirmed by the inoculation of guinea-pigs.

Metabolimetry in Hyperthyreoidism.

H. R. HARROWER (*Medical Record*, June 11, 1921) has discussed the value of estimations of basal metabolism or heat production in patients suffering from exophthalmic goitre. A rise in the basal metabolism is one of the most characteristic features of overactivity of the thyreoid gland. According to Du Bois, increased metabolism is the chief symptom of hyperthyreoidism and the determination of the heat production affords the best index of the severity of the disease. Measurements made with the Benedict calorimeter indicate that in hyperthyreoidism there is an increase of 25% to 75% above the normal heat production. In severe cases the warmth of the skin and the sweating can be accounted for by the necessity for the increased elimination of heat. Before an estimation of the basal metabolism is made, the patient is starved overnight for twelve to fifteen hours, so that food metabolism may be eliminated, and he rests in the recumbent posture for thirty minutes to one hour in order that the metabolism of voluntary muscular effort may be eliminated. The study of the basal metabolism has been of value in Graves's disease, but is not necessary either for the diagnosis or prognosis, a clinical estimation of the symptoms and signs being still the best guide for these purposes.

NEUROLOGY.

The Pathogenesis of Disseminated Sclerosis.

J. L. BIRLEY AND LEONARD S. DUDGEON (*Brain*, July, 1921) recognize two types of disseminated sclerosis. The first is the remittent type. It is characterized by acute exacerbations at widely varying intervals, alternating with quiescent periods. The second is the chronic progressive type. In the authors' series (thirty-five patients) the proportion of remittent to chronic progressive cases was as six to one. In early cases of the remittent type, once the acute disturbance has subsided, the patient may offer no clinical evidence of organic disease over prolonged periods. The possibility of spontaneous cure cannot therefore be entirely denied. The remittent type in its later stages tends to assume the characters of the chronic progressive type. The bulk of clinical and pathological evidence is opposed to the view that these two types correspond to two different pathological processes. On the contrary, they are to be regarded as manifestations of one and the same disease, namely, disseminated sclerosis. Cultural and microscopic investigation of the cerebro-spinal fluid has, in the experience of the writers, thrown no light on the pathogenesis of tne disease and no specific organism has been isolated. Their attempts to transmit disseminated sclerosis from man to animals (rabbits) have been unsuccessful. They regard the transmissibility of the disease from man to animals as unproven. They think that the evidence in favour of the assumption that the pathogenic agent is a spirochæte is incomplete and in many respects unsatisfactory; further, that the origin and nature of the morbid agent must for the present remain *sub judice*; and, finally, that the clinical and histological evidence is overwhelmingly in favour of the view that the morbid process underlying the disease is inflammatory in character.

W. E. GYE (*Brain*, July, 1921) claims that the history of the experimental study of disseminated sclerosis begins with the publication of a paper by himself in 1913. In this paper he reported the occurrence of paralysis in rabbits after injection of cerebro-spinal fluid from a patient suffering from a rapidly progressing type of the disease. He adds that these results have been confirmed by Kuhn and Steiner, Simons, Marinesco, Rothfeld, Freund and Hornowski. During the past twelve months Gye has injected 129 rabbits and 15 guinea-pigs with cerebro-spinal fluid from 21 patients suffering from disseminated sclerosis. The guinea-pigs remained healthy, but 17 rabbits became ill and paralysed. The existence in the cerebro-spinal fluid in cases of disseminated sclerosis of a living virus pathogenic to rabbits is assumed. "It would be rash, however, to draw this conclusion from such a small number of experiments without proper controls." In the affected rabbits there were no specific naked eye findings. To explain the infrequency of successful experiments it is supposed that the organism, if there be one, is not constantly present in cerebro-spinal fluid and is never present in large numbers.

Agrammatism.

O. MAAS (*Neurologisches Centralblatt*, March, 1920) points out that the difficult subject of aphasia is not made less difficult by the differences of opinion as to the exact definition of agrammatism, which is a clinical feature of a large number of cases in the aphasia group. The author follows Pick and Kleist in believing that the essence of agrammatism is inability to form sentences correctly and that defects in conjugation, declension, etc., should be called pseudo-agrammatism. He quotes cases to show that agrammatism in the stricter sense is the result of lesions of the temporal lobe. In the first case a man of 51 years presented the condition in characteristic form. After death an enormous lesion was found in the left cerebral hemisphere, undoubtedly cutting off to a large extent communication between the speech area and corresponding parts of the right hemisphere. In the second case, a man of 36 years showed that form of agrammatism known as the "telegram style" of speech. The lesion was a large cyst occupying the greater part of the left temporal lobe and the lower half of the central gyri. In both these cases a phase of practically complete aphasia had been followed by considerable return of speech, both on the motor and on the receptive side, leaving the agrammatism as above mentioned. Very briefly, the author's main conclusion is that there is no "centre," a lesion of which will produce agrammatism, but that this condition is the result of an attempt on the part of the right hemisphere to assume speech functions as a whole when it is less well equipped for so doing than the left hemisphere.

Froin's Syndrome.

J. G. GREENFIELD (*Journal or Neurology and Psychopathology*, June, 1921) summarizes what has been previously written on Froin's syndrome and records a striking series of cases. The syndrome concerns a change in the cerebro-spinal fluid which, upon being received into a test tube in the usual way, may be completely coagulated and present a yellow colour. The syndrome consists essentially in the approximation of the character of the fluid to that of blood plasma. The change occurs when the fluid in the lumbar *cul de sac* is cut off from communication with the fluid in the ventricles and *cisterna magna*. This may be produced by tumours or other disease in the bones of the spine, by tumours of the meninges or cord or by inflammatory adhesions in the pia-arachnoid membranes. The degree of change in the fluid depends more on the completeness of this block than on the nature of the blocking process. But certain constituents may vary in relation to the nature of the obstruction. The production of the syndrome is aided by venous congestion below the level of a compression, or by inflammation in the meninges and cord below an area of meningeal adhesion. It is not necessary to postulate any obstruction of the peri-neural or perivascular lymphatics. The lymph, which reaches the subarachnoid space along them, aids in the production of the syndrome. In fact, acute peripheral neuritis may itself produce an analogous condition in the cerebro-spinal fluid.

Torsion Spasm (Syndrome of the Corpus Striatum).

A. WIMMER (*Revue Neurologique*, Nos. 9-10, 1921) records an interesting case of torsion spasm. A girl of twelve years, in whose family none was similarly affected and who showed no sign of congenital syphilis, had suffered for two years from a slowly progressive affection of the nervous system, characterized by choreiform movements and involuntary contortions affecting almost every muscle of the body, giving rise to most extraordinary attitudes and not ceasing during sleep. These movements, which dominated the clinical picture, were accompanied by speech disturbance alone. On the negative side, muscular rigidity, paresis, increased tendon reflexes, the sign of Babinski, sphincter weakness, sensory disturbance, oculo-motor paralysis, disc change, nystagmus and mental impairment were all absent. At the autopsy the *corpus striatum* in each hemisphere was found to be gravely degenerated and the liver presented a cirrhotic appearance. So far the lesions corresponded with those of Wilson's disease. There was this difference, however, in addition to the affection of the *corpora striata*, there were scattered sclerotic changes in various parts of the brain.

The Abdominal Crises of Migraine.

J. ARTHUR BUCHANAN (*The Journal of Nervous and Mental Disease*, November, 1921) states that attacks of abdominal pain occasionally occur as a radical of the manifestations of migraine. Such attacks are periodic, sudden in onset, deep-seated in the epigastric region and associated with a feeling of pressure or fullness, with eructations and sometimes with vomiting. Buchanan reports seven cases observed in patients at the Mayo Clinic and points out that five of these had been operated upon for abdominal trouble, though exhaustive examination had failed to reveal any abnormality. He emphasizes the futility of surgical interference and the importance of studying the family tree for evidence of migraine. Migraine, he adds, has always been considered hereditary, but it is so only to a certain extent, not at the rate 100%, but in a simple Mendelian way. He also thinks that migraine needs to be taught, not as a disease, but as a special type of a normal condition.

British Medical Association News.

SCIENTIFIC.

A MEETING of the South Australian Branch was held in the Lister Hall, B.M.A. Buildings, Hindmarsh Square, Adelaide, on October 27, 1921, the President, DR. BRONTE SMEATON, in the chair.

Extensive Wound of the Thorax.

DR. H. A. POWELL, C.M.G., exhibited a patient who had sustained extensive injuries of the right side of the thorax as a result of wounds received in the great war.

Transposition of the Viscera.

DR. W. R. CAVENAGH MAINWARING showed a boy in whom the viscera were completely transposed. He also demonstrated skiagrams illustrating the condition.

Osteo-Sarcoma of the Femur.

PROFESSOR J. B. CLELAND demonstrated on behalf of DR. CAVENAGH MAINWARING, DR. D. L. BARLOW and himself certain pathological specimens and sections of a case of osteo-sarcoma of the femur extending to the tibia.

The patient from whom the specimens had been removed by operation, was a boy of fifteen years. The interesting features of the case were the previous history of repeated mild trauma, the great size of the growth, the extensive periosteal involvement of the femur and the similar but less extensive periosteal extension to the tibia without gross changes in the bone.

The case history showed that the boy, who had been admitted to the Adelaide Hospital on October 4, 1921, had noticed a definite swelling in the vicinity of the left knee joint during the previous twelve months. He could recall no definite injury sustained by the limb, but suggested that the pressure of a garden pitch-fork used for the tossing of hay into a waggon might have caused the swelling. He stated that the swelling was slight at first and had been treated with fomentations. As it became gradually larger, the swelling had been incised and the wound plugged. The tumour had then begun to increase rapidly in size. On examination, the boy showed obvious signs of loss of weight. He was pale and there was considerable wasting of his thoracic and abdominal muscles. The left knee joint was swollen to the size of a large punching ball. It was hot on palpation and the skin covering the swelling was shiny and traversed by distended venules. The tumour occupied the greater portion of the femur, extending from the proximal third downwards and at the same time gradually increasing in size and assuming a more or less oval shape in the vicinity of the knee joint.

The tibia appeared to be only slightly involved in the growth and the fibula to escape completely. The femur was the structure chiefly involved and the growth was hard and bony in character. On the surface of the swelling the old operation scar stood out prominently.

Skiagrams of the lower limb showed an extensive bony tumour involving the soft parts of the lower half of the femur. It extended down to, but apparently was not growing from, the tibia. There was a marked periosteal reaction up the shaft of the femur. The appearances were those of a periosteal sarcoma. Skiagrams of the hip joints showed indefinite changes. An irregularity of the fifth lumbar vertebra and an obliteration of the left sacro-iliac synchrondrosis suggested the possibility of a secondary deposit affecting this region. No other spine irregularity was noted. Skiagrams of the lungs revealed certain small rounded opacities in the upper part of the left hilum which were possibly small metastatic growths. Apart from these changes there was no evidence of metastasis.

The operation of disarticulation at the hip joint was carried out on October 14, 1921. The patient became almost pulseless near the completion of the operation, but later revived. He was given rectal injections of saline solution and brandy and intravenous injection of saline solution with gum arabic.

Since the operation he had shown marked improvement.

Macroscopic examination of the specimens showed that the lower two-thirds of the thigh and the region of the knee joint were involved in a firm swelling, the transverse diameter of which was 16.25 centimetres. The antero-posterior diameter measured 17.5 centimetres. Section of the mass revealed a very large ossified new growth involving the lower two-thirds of the femur. The muscles and soft tissues which remained formed only a thin layer stretched over this growth. At the lower end of the femur the growth extended slightly through the articular cartilage of the inner condyle into the knee joint and an irregular extension of osseous new growth behind the head of the tibia was also apparent.

The original shaft of the femur was distinguished with difficulty towards the posterior part of the growth, except at the upper end, where it tapered off along the bone. The medullary canal contained osseous material throughout the affected area. The growth contained irregular calcified trabeculæ, pale in colour, with very small areas of soft tissue in between. The new growth, as it ascended the femur, appeared to involve the periosteum alone. It was thick below where it joined the main mass and gradually became attenuated above, till it disappeared high up the shaft. A similar periosteal involvement affected the upper few centimetres of the tibia, with a similar attenuation downwards. The actual cavity of the knee joint was hardly involved at all, the growth having "jumped" the joint to reach the tibia.

Examination under the microscope of sections of the growing part of the tumour revealed in the older and deeper portions numerous narrow bony trabeculæ separated by cellular sarcomatous tissue. Towards the periphery the cellular areas became more extensive and the bony formation became less, whilst areas of incipient osteoid tissue were visible. In its earliest stage the osteoid tissue appeared as a granular transformation of the matrix around a cell or between the members of a group of cells. This area underwent enlargement, so that each cell became widely separated from its fellows. The osteoid tissue extended and later underwent calcification into irregular osseous trabeculæ, which anastomosed more or less with each other. The sarcoma cells were moderately large and irregularly polygonal in shape. Many of them were characterized by large nuclei and prominent nucleoli. They lay rather close together in a slightly reticular matrix, except when separated by the early formation of osteoid tissue. In the osteoid and osseous tissue, the enclosed cells at first large and irregular. Many of them contained short processes and lay in lacunæ. The older cells became shrunken. The surrounding cells became pushed aside and those nearest the osteoid tissue became elongated and attached to that tissue, as though they were plastered on to it. Further, osteoid transformation could be observed around the osseous strands, leading to their increase in thickness, with corresponding diminution of the sarcomatous areas between. The cells had become more or less atrophic. In older parts the tissues stained diffusely with eosin. Osseous trabeculæ were seen widely separated by apparently an indefinite, granular, calcified matrix containing scattered degenerating cells. In the matrix and in the osseous trabeculæ shrunken nuclei could be seen lying in swollen, clearer spaces, presenting an appearance suggestive of cartilage.

Hypertrophic Pulmonary Osteo-Arthropathy.

DR. HELEN MAYO showed a child suffering from hypertrophic pulmonary osteo-arthroplasty. She also exhibited skiagrams of the child's hands, feet and thorax.

Sarcoma of the Uterus.

DR. HELEN MAYO then read notes and exhibited a pathological specimen of a case of sarcoma of the uterus. The morbid anatomy and microscopical appearances of this specimen were described by Professor J. B. Cleland and Dr. D. L. Barlow.

The patient was a multipara, sixty-nine years of age, in whom the menopause had occurred eighteen years previously. She gave a history of a blood-stained, slightly offensive discharge during the preceding two weeks. On examination, the bleeding was observed to be of uterine origin and the uterus was found to be enlarged. She was subjected to operation three weeks later.

The enlargement of the uterus was found to be due to the presence of a tumour of about the size of a hen's egg.

It had the appearance of a myoma which had undergone degeneration. A slender pedicle ran from the tumour, pierced the endometrium and formed a button-like vascular projection inside the uterus. This was of interest since there was no means of proving the malignancy of the growth prior to operation. Curettage failed to remove the button-like growth. But even if it had been possible to remove portions of the growth by curettage, the pathological findings would not have been definite enough to prove malignancy. The patient improved greatly and left the hospital within three weeks.

Examination of the pathological specimen showed that a growth, five centimetres long and 3.75 centimetres wide, was present in the anterior wall of the uterus. Section of the specimen revealed in its upper part a yellowish caseo-calcareous portion with scattered hæmorrhagic areas, in the lower part a fleshy, greyish-pink portion with well-marked vessels, the appearances suggesting a degenerated fibro-myoma. Near this growth were several small, partly calcified fibro-myomata. In the uterine mucosa, opposite the middle of the large growth, was a small vascular polypoid projection, nine millimetres by six millimetres in size, with a constricted base. It was connected by a distinct greyish-pink core through the uterine wall with the large tumour.

Microscopically the large growth showed numerous moderately large cells lying in a fine reticulum. The cells apparently contributed to the reticulum by means of numerous processes. The nuclei were large, sometimes extremely large. Occasionally there were two to each cell. In places remains of the original fibrous stroma could be seen and here the connective tissue cells were very large and contained processes and large nuclei. In places old blood pigment was present in the cells. The projecting part of the polypus showed large vascular spaces with indefinite walls embedded in a cellular stroma. Hæmorrhages were present in places. The matrix, apparently fibrous, became more cellular as the core was approached, the cells being medium-sized and irregular. There were several large vascular spaces lined by endothelium and some smaller capillaries. In places the original dense fibrous strands showed between them bands of irregular cells in a matrix, usually accompanied by capillaries. The growth was either a sarcoma or else a cellular fibroma. Mitotic figures were few. The connexion of the polypoid projection with the neoplasm suggested infiltration, a point in favour of malignancy.

Exhibit of Marshall's Portable Gas-Oxygen Apparatus.

DR. GILBERT BROWN exhibited Marshall's portable gas-oxygen apparatus. He demonstrated the simple sight-feed, by which the proportion of the gases could be accurately measured. Each of the gases passed to a metal tube which dipped into water. The metal tubes were open at the lower end and had side holes at intervals of 1.25 centimetres above. As the pressure was increased, the gas bubbled through more and more holes or even through the end of the tube. When anæsthesia had been established, it could usually be continued with "four holes" of nitrous oxide and "one hole" of oxygen. The depth of anæsthesia might be increased by increasing the percentage of nitrous oxide, by re-breathing and by adding ether. The addition of ether could be effected by opening a by-pass so that the mixed gases bubbled through the ether.

The advantages of gas-oxygen anæsthesia for major operations were stated to be the rapid induction of two to three and a half minutes, the rapid recovery, which took place almost immediately after the face piece was removed, the great diminution of shock and the absence of post-anæsthetic nausea and vomiting.

The disadvantages were the high cost of the gases (for an operation of forty to sixty minutes' duration the cost was approximately £1), the weight of the cylinders and apparatus and the consequent decreased portability and the occasional failure to obtain complete relaxation of the patient.

Pathological Report of a Case of Pulmonary Tuberculosis.

PROFESSOR J. B. CLELAND described a case of fibrosing pulmonary tuberculosis. The patient showed *post mortem* evidence of very old, probably infantile and bovine tuberculosis of the mesenteric glands, which ended in complete recovery with calcification of the caseated areas. Presumably many years afterwards, a fresh human tuberculous infection occurred. The patient had had half of his tongue removed eight years prior to his death for epithelioma, when presumably he showed no signs of pulmonary disease. Had the progress of this second infection been modified at all by the previous bovine infection? If not, could it be reasonably concluded that tuberculin would have had any effect? In Professor Cleland's opinion, the course of events had been modified. The lungs showed considerable fibrosis with what might be called a fibrosing tuberculous peri-bronchitis. Such a fibrosing condition indicated a good response on the part of the tissues against the invasion by tubercle basilli, which was probably the chief, if not the only, way by which a tuberculous process could be cured or relatively controlled. Another interesting feature was the unusual degree of hypertrophic tuberculous granulomatosis affecting the base of the tongue, the adjoining parts of the pharynx, the epiglottis and the neighbouring tissues almost as far down as the false vocal cords. It was an unusual condition. There appeared to be an increase of the lymphoid tissue. In addition, there were an extensive infiltration with plasma cells and proliferation of the fixed cells and endothelial cells. Though tubercle bacilli were numerous in places in the granulomatous tissue over the epiglottis, giant cells and tubercles did not appear. A few giant cell systems were seen, however, at the base of the tongue. If the hypertrophic reaction—in contrast to an ulcerative lesion—also indicated increased resistance to the invasion, it was of a different order to the fibrosis which was visible in the lung. Doubtless that part had been infected from the lung, as had the Peyer's patches in the intestine, which showed no special indications of increased resistance. Was it right to assume that, had this patient not had a previous infection of his mesenteric glands, his fresh pulmonary disease would have assumed a more rapidly progressive form than did actually develope? If that could not be inferred, could it be supposed that tuberculin injections would have modified matters?

Professor Cleland then described the clinical features of the case: The patient was a male, aged sixty-nine years. Eight years previously half of the tongue had been removed on account of an epitheliomatous growth. He had had no further trouble from this source. After the lapse of several months he began to have dysphagia and cough and commenced to lose weight and strength. Tubercle bacilli were found in the sputum and since admission to hospital wide-spread signs had developed in the chest.

At the *post mortem* examination, universal fibrous adhesions were found over both lungs. Almost the whole of the upper lobe of the left lung showed considerable fibrosis, with caseated areas and cavities up to the size of small marbles; some air-containing tissue remained in the lower part of that lobe. The lower lobe presented a number of racemose clusters of tubercles along the course of the bronchioles. The right lung also showed in the upper lobe racemose clusters of small tubercles, with considerable fibrotic reaction, giving the appearance of a network; isolated tubercles appeared in the lower part of the upper lobe. The middle lobe showed scattered clusters of tubercles. The lower lobe was rather thickly studded with little racemose clusters of tubercles, the larger becoming caseated. The lymphatic tissue at the base of the tongue was hypertrophied and appeared as low, firm, irregular, nodular projections. The epiglottis was also thickened and irregularly nodular and presented on the left side near the tip a small ulcer. The mucosa of the larynx down to but not including the true vocal cords was also affected. There were typical tubercular ulcers in the ileum. There were old calcified tubercular glands in the mesentery near the caecum.

Discussion on Pulmonary Tuberculosis.

A discussion then took place on the paper by DR. J. WALTER BROWNE, "The Prevention and Treatment of Pulmonary Tuberculosis," and on that by Dr. D. R. W. COWAN, "The Problem of Tuberculosis," both of which were read at a meeting of the Branch on August 25, 1921 (see THE

MEDICAL JOURNAL OF AUSTRALIA, October 22, 1921, pages 333-347).

DR. A. A. LENDON congratulated Dr. Browne and Dr. Cowan on the merits of their papers. He said that the President would doubtless recall those dark days of the nineteenth century when general practitioners like themselves ventured to diagnose and were permitted to treat pulmonary tuberculosis. Now they recommended tuberculous patients to the care of a specialist. He was not complaining, but merely recording a fact. He had been impressed by the difference in the views expressed in the two papers under discussion. He took comfort from the fact that the opinions of Dr. Browne and himself seemed to coincide in the view that the tuberculin treatment had not made good. Dr. Browne's discourse seemed to him philosophic and dispassionate. Dr. Cowan's paper, on the other hand, was more vehement and impulsive. Dr. Cowan was certainly less tolerant of such weaker brethren as Sir J. C. Verco and Dr. Hayward. He (Dr. Lendon) felt quite uncomfortable when he reflected how many cases of dyspepsia had missed the saving grace of a von Pirquet or other tuberculin test. A night's sleep and reflection, however, had restored to him his normal mental equilibrium. He began to recall some of his cases. He thought of a man sent home to England to die, who found his health on the old sailing ship *Torrens*, which was almost saturated with tubercle bacilli. He thought of a lady condemned to die at twenty-five, whose death certificate he had signed at the age of sixty-three. He recalled a patient who recovered after "roughing it" for three months on the back-blocks of the Murray, whilst his brother also recovered and had not been absent one day from his office in the city. He recalled patients who had improved just as much with open-air treatment at Gilberton, on the bank of the river, as at Mount Lofty. The fact was that all forms of treatment might be followed by benefit and all might be lamentable failures. He had even known Alabone's treatment to be successful.

Some time ago urea had been vaunted as the great cure of tuberculosis, then guaiacol, then chloramine-T. His own favourite remedy was the succinimide of mercury. He had obtained good results from the use of it and he had also experienced failures. Increase of weight alone was not a satisfactory test of a cure. A patient of his had returned from a sanatorium in England bloated almost beyond recognition, but neither her chest nor larynx had improved. Marcus Patterson's book, "The Shibboleths of Tuberculosis," was well worth reading, though it was doubtful whether he had proved his case with regard to auto-inoculation.

DR. F. S. HONE stated that he had, like Dr. Lendon, changed his method of treatment of pulmonary tuberculosis from time to time. He was in thorough agreement with Dr. Cowan in regard to the importance of early diagnosis. The papers illustrated the danger of praising any particular form of treatment to excess. In connexion with the subjection of prevention, he was at variance, not only with what Dr. Browne had said, but also with the complacency of Dr. Browne's policy of despair. He (Dr. Hone) had been impressed by the fact that authorities all over the world were urging investigation into the methods of prevention of the disease, apart from the question of prophylactic inoculation. Dr. Browne's statement that infection did not occur with facility in adults was not supported by the statistics of conjugal infection. No physician was as yet conversant with the conditions which determined the spread of infection of pulmonary tuberculosis in Australia and no one had the right to dogmatize. The figures usually quoted were those of thickly populated countries. An investigation of Australian conditions was necessary.

He disagreed with Dr. Browne's statement that the mortality rate of pulmonary tuberculosis was falling. The fact was that since 1910 the death rate had risen. He also differed from Dr. Browne's statement that it was seldom possible to trace the source of infection to a second person. He believed that close contact and massiveness of infection were extremely important factors in the spread of the disease. It was important that all should strive to be proficient in the diagnosis of incipient disease and in the education of infected persons.

SIR JOSEPH VERCO stated that he was convinced that one adult could infect another with pulmonary tuberculosis. He had been astonished to find how one method of treatment after another had been claimed to be curative and then, after a time, dropped and he was of opinion that this fact should make medical men cautious in claiming the discovery of remedies. After several years' treatment with tuberculin, the medical profession as a whole was not persuaded that tuberculin was a cure for tuberculosis. He was quite certain that prophylaxis should be the aim of the profession. If milk infected children, milk infection could be and should be prevented. If segregation were necessary, segregation should be insisted on. Those were the methods by which the disease should be attacked.

DR. W. T. HAYWARD, C.M.G., said that doubtless all medical men who had been in practice for many years, could recall cases of pulmonary tuberculosis in patients who were apparently incurable but who had nevertheless recovered. Moreover, it was well known that *post mortem* examinations frequently revealed scars of tuberculous lesions which much have been active at one time but had since completely healed. How was it, then, that the treatment of the disease was so very unsatisfactory? He thought the answer was that the diagnosis was too often delayed till the physical signs in the lungs or the presence of tubercle bacilli in the sputum made it certain. The disease must have reached a rather advanced stage before such manifestatinos could occur. Up till comparatively recent time the condition of the heart in cardiac diseases had been chiefly gauged by the physical signs detected by the stethoscope. Thanks to Sir James Mackenzie, it was now recognized that symptoms gave earlier and more reliable information. He (Dr. Hayward) considered that it was only by adopting a similar viewpoint for the diagnosis of pulmonary tuberculosis, namely, by the more efficient study of the symptomatology of the disease, that it would be possible to hope for any greater success in combating it. The symptoms were those due to the toxæmia. He thought it more than probable that the use of tuberculin for diagnostic purposes would be of great value in the early recognition of the disease. He could only hope that it would be equally valuable for its treatment.

DR. HELEN MAYO called attention to the importance of common colds and respiratory disturbances as antecedents to tuberculous infections. Referring to a comparison which had been made between the tuberculin test and the Wassermann reaction, she pointed out that, whereas the Wassermann test was innocuous to the patient, there was the distinct danger that the use of a diagnostic injection of tuberculin might "light up" a latent tuberculous focus.

DR. GLEN H. BURNELL strongly supported Dr. Cowan in regard to the use of tuberculin for diagnostic purposes. He believed that a better tuberculin for this purpose would still be found. In his opinion, those practitioners who had condemned the use of tuberculin, had probably used it very little. If practitioners would use tuberculin without prejudice as a diagnostic agent and record their results over a period of several years, some finality as to its utility might be reached, but not until this was done could the subject be discussed among a similar gathering of practitioners with intelligence.

DR. J. W. BROWNE, in his reply, stated that the subject was so large that he had found it impossible to touch on all points in the paper which he had read. He thought that both vaccine therapy and chemio-therapy had their good results, but it was impossible to say whether any instance of cure or arrest was due to any particular part of the treatment. He preferred auto-intoxication to the injection of a general toxin. He had meant his paper to be stimulating and if it had stimulated discussion he was satisfied. Probably owing to the condensation of his paper, his remarks on the subject of a filtrable virus had been misunderstood. He thought that the conjugal infectivity of husband and wife was a disputed point. The wives of grinders at Sheffield were not more subject to tuberculosis than the wives of other men. With regard to the mortality rate, investigations had not been fully prosecuted in Australia and his remark concerning this subject was a general statement. He agreed with Sir Joseph Verco that there had been no progress in the treatment of the

disease. If treatment were commenced at an early stage, a cure might be obtained, but at a late stage the result was very doubtful. At the Kalyra Sanatorium for the year ended June 30, 1921, the results of cases treated were as follows: Of thirty-four patients in the first stage, thirty had the disease arrested. Of thirty-nine in the second stage, the disease was arrested in nine and of twelve in the third stage, the disease was arrested in one. He agreed with Dr. Hayward in regard to the importance of studying the early stages of the disease.

Professor Wassermann and other scientists had recently stated their opinion that no tuberculin could be found which would cure tuberculosis, for the reason that tuberculosis in man apparently gave no immunity to the person infected such as was given by small-pox, scarlet fever and many other infections. Therefore, in expecting a vaccine to bring about an absolute cure in tuberculosis, physicians were asking more than even Nature granted. He preferred to utilize the patient's own "tuberculin."

DR. D. R. W. COWAN replied briefly. He thought that an early diagnosis of tuberculosis could not be definitely made without the use of tuberculin. He would like to see more medical men using tuberculin for that purpose. When tuberculin was more freely used a definite verdict as to its clinical value might be pronounced.

A MEETING of the South Australian Branch of the British Medical Association was held on November 24, 1921, at the Lister Hall, Hindmarsh Square, Adelaide, DR. JOHN CORBIN, in the absence of the President, in the chair.

Hymenolepis Diminuta.

DR. GLEN H. BURNELL exhibited a specimen of *Hymenolepis diminuta* recovered from a child.

Gun Shot Wounds.

DR. JOHN CORBIN showed a series of skiagrams to illustrate the condition of three patients under his care at the Repatriation Hospital and read notes concerning these patients.

The first patient was 25 years of age. He had received a penetrating wound in the right side of the chest in September, 1917. He had hæmoptysis, but the foreign body had not been removed. There has been a discharge from the wound at first, but within a month the wound was healed. The patient had had no other wounds or sickness while abroad. Since his return to Australia he had been doing light work. He had suffered some pain in the chest. In December, 1920, there had been an attack of severe pain in the chest, accompanied by slight hæmoptysis. He had been confined to bed for four days.

Late in June, 1921, his chest affection had become troublesome. While walking in the street he had had an attack said to have been accompanied by unconsciousness. When seen he appeared to have been in severe pain, gasping for breath and tearing at the right side of his chest. Morphine had no noticeable effect. The attack lasted for three hours and recurred at intervals. After ten days he had been sent to the Keswick Hospital.

The patient did not sleep for two days, despite morphine. He complained of constant and severe pain. He had a cough of the pleural irritation type. Some relief was obtained by tight strapping. Examination of the chest disclosed some diminished breathing at the base of the right lung in front, with some impairment of the percussion note. No tubercle bacilli were found in the sputum. There was a slight chronic pharyngitis.

On August 24, 1921, Dr. H. C. Nott examined the patient radiographically. He reported that a foreign body could be seen lying behind the chest wall. On screen examination it was found that the foreign body moved downwards with each excursion of the diaphragm to the extent of about two centimetres. Its sharp anterior extremity then appeared to catch in some structure and the posterior end was drawn downwards through an angle of at least 90°. From these appearances Dr. Nott had assumed that the foreign body was lodged in lung tissue, with its sharp ends projecting into the pleural sac and catching in the parietal pleura during respiration. An operation was carried out two days later. An incision was made in the anterior chest wall on the right side and a portion of the sixth rib with its cartilage was removed. The pleura was opened and the foreign body was found in the lung tissue lying immediately below it. The foreign body was removed. Recovery was uncomplicated and no further pain was experienced. The patient's weight had increased by five kilograms since the operation.

The second patient was aged twenty-three years. He had received a wound in the pelvis on October 17, 1917. The foreign body had not been removed. A small sinus had formed in the right groin, which had healed some months later. The sinus had not broken down. The patient had suffered occasional pain after micturition and there was some frequency of urination up to August, 1920. He then became ill with fever and pain. After seven days a large quantity of pus was discharged in the urine and relief followed. A similar but less severe attack occurred three months later and three other attacks followed during the succeeding six months.

On June 11, 1921, he was admitted to the Keswick Hospital. With the exception of the presence of *Staphylococcus albus*, no bacteria were grown from the urine; the chemical analysis did not reveal any important change. On July 21, 1921, Dr. H. C. Nott reported the result of an X-ray examination. Stereoscopic skiagrams had been taken with a silver catheter in position. A foreign body was seen about three centimetres to the right of the *symphysis pubis*, at the level of the bone and between one and a half and two and a half centimetres behind the upper border of the ascending ramus of the pubis. Dr. Nott considered that the foreign body was lying very close to, if not actually within, the wall of the bladder.

An operation had been performed at a later date. The incision had been made in the middle line above the pubes and the bladder fixed by means of sutures and carefully examined. No foreign body had been detected from outside. The bladder had then been opened and a patch of scar tissue had been discovered above the right ureteric orifice. The foreign body had been found below this scar tissue and had been removed. The patient made an uninterrupted recovery, except for a single hæmorrhage into the peri-vesical region.

The third patient had had a gun shot wound in the neck in April, 1918. The foreign body had not been removed. A sinus had appeared in the right side of the neck and had persisted. Skiagrams taken in 1920 had revealed a large piece of metal lying in close relation to the second and third cervical vertebræ. The sinus had been enlarged and an unsuccessful attempt had been made to remove the foreign body.

In September, 1921, the patient had come under Dr. Corbin's care on account of cellulitis of the neck. The swelling had been opened and much pus had been discharged. The inflammation had subsided and a small sinus had persisted just behind the *sterno-cleido-mastoideus* muscle. A skiagram taken on September 6, 1921, had revealed a large piece of metal lying apparently in the space between the second and third cervical vertebræ on the right side. Dr. Nott had expressed some doubt as to the existence of an osteitis in the region of the inter-vertebral articulation. On October 27, 1921, a vertical incision had been made to the right of the middle line and the second, third and fourth cervical vertebræ had been exposed. No foreign body had been found, notwithstanding a prolonged search. The bone had not appeared to be much thickened. The wound had therefore been closed. It had healed by first intention. On November 9, 1921, Dr. Nott had carried out a stereoscopic X-ray examination. He had found that the atlas had been fractured apparently in the region of the right articular process. The inferior limb of the process had disappeared. The foreign body causing the damage had apparently been deflected downwards and had been arrested at the spinal foramen against the lamina of the third cervical vertebræ.

Dr. Corbin explained that the object of showing the skiagrams and of reading the notes was to illustrate the importance of stereoscopic X-ray examination in localizing foreign bodies.

DR. H. CAREW NOTT explained the method used in obtaining stereoscopic views.

Malignant Cyst of the Ovaries.

DR. A. A. LENDON read the notes of a malignant ovarian cyst with splenic, peritoneal and pleural metastases (see page 323). He also read the pathological notes by DR. C. T. C. DE CRESPIGNY (see page 325).

DR. W. T. HAYWARD, C.M.G., said that Dr. Lendon's paper recalled to his mind the case of a patient who had come under his care some ten years previously. Dr. Cudmore had operated on this patient. When the abdomen had been opened, large quantities of colloid material had escaped. Dr. Hayward was not in a position to state whether the pathology was the same as in Dr. Lendon's patient.

Melæna Neonatorum.

DR. BRIAN H. SWIFT, M.C., read a paper entitled "Transfusion With Small Amounts of Mother's Blood in Melæna Neonatorum" (see THE MEDICAL JOURNAL OF AUSTRALIA, November 26, 1921, page 482). A discussion followed.

TRANSACTIONS OF THE COUNCIL OF THE VICTORIAN BRANCH.

THE following is a summary of the more important transactions of the Council of the Victorian Branch of the British Medical Association during January and February, 1922:

Patients in Public Hospitals.

The Council adopted the following resolution:

> That an honorary medical officer of a hospital should refrain from attending discharged patients, except as a consultant, unless he is satisfied there was no previous medical attendant.

Leave of Absence.

Leave of absence was granted to DR. J. W. DUNBAR HOOPER and DR. J. F. WILKINSON, who were leaving for extended holidays in Europe. The former was entertained at dinner at the Oriental Hotel on Saturday evening, March 4, 1922, when over sixty of his colleagues met to bid him *bon voyage*.

Manchester Unity Order of Oddfellows.

Representatives of the Manchester Unity Order of Oddfellows met the Organization Committee and asked the Committee to recommend the medical profession in Victoria to accept lists of members and to be paid at the rate of 10s. 6d. for the first visit, 7s. 6d. for the second and 5s. for subsequent visits. They could not agree as a matter of principle to the income limit clause of the Wasley Award. The request was declined.

Medical Agency.

It was decided to adopt the following objects of the Agency:

(*a*) To promote fair dealing between buyer and seller.
(*b*) To supply thoroughly reliable *locum tenentes*.
(*c*) To undertake such business concerns as may from time to time meet with the approval of the Medical Society of Victoria.
(*d*) To provide a fund to meet the expenses of the Library, for clerical assistance and for such extraordinary expenditure as cannot be conveniently met from the funds of the Victorian Branch of the British Medical Association or from those of the Medical Society of Victoria.

Order of St. Andrew.

Delegates met the Interpretation Committee and asked that the Order of St. Andrew should be permitted to sign an agreement with the Victorian Branch of the British Medical Association, although one lodge only, consisting of eight members, refused to come into line. The request was refused.

"On" and "Off" Certificates of Lodges.

At the request of the Victorian Branch of the British Medical Association, the Friendly Societies' Association has provided uniform certificates, which have greatly simplified the work of the medical officer. The Ancient Order of Foresters is not a member of the Association and declines to pay its proportion, although the medical officers make use of these certificates for Ancient Order of Foresters lodge members. The Council asks lodge medical officers to refrain from using these uniform certificates for Ancient Order of Foresters work and apply immediately to the local secretaries of the Ancient Order of Foresters for a supply of Ancient Order of Foresters certificates.

Patients in Public Hospitals.

Practitioners sending patients to public hospitals are now able to learn the progress of the case, the prognosis and treatment advised after discharge. A printed list of questions has been drawn up by the Council and the staffs of the public hospitals have agreed to supply answers whenever application is made upon the prescribed form. Copies of these questions may be obtained from the Secretary of the Victorian Branch of the British Medical Association and a charge of 1s. 6d. per dozen is made to cover the cost thereof.

NOTICES.

APPROVAL having been given by a general meeting of the Victorian Branch of the British Medical Association to the formation of a Section of Neurology and Psychiatry, it is requested that all those interested will forward their names without delay to DR. R. C. WITHINGTON, 83, St. George's Road, Elsternwick, or to DR. PAUL DANE, 80, Collins Street, Melbourne.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

HALES, GEOFFREY MAURICE BARNEWALL, M.B., Mast. Surg., 1921 (Univ. Sydney), Royal Alexandra Hospital for Children, Camperdown.

WIPPELL, WALTER PLANTAGENET, M.B., Ch.M., 1921 (Univ. Sydney), Royal South Sydney Hospital, Zetland.

THE AUSTRALIAN MUSEUM, SYDNEY.

WE have beeen asked to announce that the programme of popular science lectures of the Australian Museum, Sydney, includes the following items:

"The Extermination of Vermin," by DR. J. S. PURDY, D.S.O., on March 22, 1922.
"The Story of the Hookworm," by DR. W. A. SAWYER, on April 13, 1922.
"Life in Past Ages," by C. ANDERSON, ESQ., M.A., D.Sc., on May 11, 1922.

Correspondence.

THE OCCURRENCE OF HYMENOLEPIS DIMINUTA AS A HUMAN PARASITE IN AUSTRALIA.

SIR: In your issue of March 4, 1922, there is an article by Dr. Burnell, of Adelaide, on *Hymenolepis diminuta*. Both Dr. Burnell and Professor Harvey Johnston overlook the fact that I made what I take to be original record for Australia of this tapeworm as a human parasite in your journal of August 7, 1920, and referred to it again in your issue of April 23, 1921. In March, 1920, I removed several complete and many incomplete specimens of this cestode from an infant of thirteen months, using fifteen minim doses of fresh extract of male fern in emulsion.

I found many rats collected in the vicinity of the bush home of this infant to be infected heavily with *Hymenolepis diminuta*.

Apart from making an original record, I was concerned to try and establish that a direct development of this cestode takes place in the human, *i.e.*, that the ovum could develope into an adult tapeworm without the advent of a second host.

Grassi and Rovelli had shown that such a development

occurs in *Hymenolepis murina* in rats and mice. The gross infection of the infant made it appear that the ingestion of a few rat droppings crowded with cestode ova was much more likely to occur than the ingestion of many fleas.

However, I could not find any other infant parasitized by this tapeworm. In view of the rarity of this parasite in humans, it must surely be looked upon as an incidental parasite of man. Yours, etc.,

CHARLES BADHAM.

Microbiological Laboratory, Department of Public Health, Sydney, March 8, 1922.

THE AUSTRALIAN PHARMACEUTICAL FORMULARY.

SIR: In your editorial entitled "Pharmacology and Therapeutics" of even date, you strike me as deserving the rebuke administered by Pope to Dr. Arbuthnot. While admitting some good points in the Australian Pharmaceutical Formulary, you say that: "If medical practitioners would study the British Pharmacopœia and would study the advances in pharmacological science, they would find less need for ready-made formulæ of prescriptions." In another place you agree with Dr. W. N. Robertson that they don't. May I ask also how many medical men have a copy of the British Pharmacopœia in their possession at the present time? More than that, how many of them have ever set eyes on the book? How many know the date of the last issue? If you can find 1% of your subscribers with the necessary proof to fulfill the above conditions I will undertake to pay the cost of printing their names in gold in the next list of members of the British Medical Association published your journal. You are ungracious to pharmacists when you "just hint a fault" in that they did not discuss "some question in therapeutics." What would you have said if they had done so in the face of your remark: "His entire lack of knowledge of therapeutics is not recognized by any layman." Mr. Cowley was meticulously careful not to express any views concerning that science and evidence to that effect is found in the printed word in the Australian Pharmaceutical Formulary. A sweping assertion that counter prescribing is wide-spread is hardly fair. A large proportion is forced on the dispenser and in almost all such cases, if it be not done, the customer will do it for himself by pointing to a glass case containing proprietary remedies much more harmful possibly than the simple *placebo* the pharmacist can dispense. What if the same gentleman adopts the "haphazard method of guessing at a diagnosis" when the best of us cannot claim to do more at times. I am not defending indiscriminate counter prescribing, but I believe that it is an act that the pharmacist will avoid whenever he can. Are not medical practitioners somewhat to blame? Not only country doctors, but city dwellers as well commonly write no prescriptions for the case referred to them by the chemist, or, if they do, they will order a full bottle of some expensive proprietary importation the retail price of which paralyses the patient (if he have not already the complaint) and leaves the unfortunate chemist with a net profit expressed in pence. Now, if you will grant that the average or more than the average practitioner cannot write a prescription based on a sound knowledge of the British Pharmacopœia, not forgetting "Martindale and Squire," then you must admit that they require ready-made formulæ. I join issue with you when you claim virtues in a secret preparation against those formulæ in the Australian Pharmaceutical Formulary made according to the methods and with the materials described in the British Pharmacopœia. The only one you mention is the arseno-benzol group. We will grant you that, if you like, but Mr. Cowley's pupils can make those preparations if put to it. However, I am not so sure that we cannot do without them, so long as we have "*Cremor hydrargyri*, A.P.F.." I have given Australian Pharmaceutical Formulary preparations a good trial since their inception and want nothing better. The advantage appertaining to their use is not confined to hospitals by any means. In private practice the patient is getting a drug in combination that I know something about, the patient is paying a reasonable price and last, and why least, the dispenser is securing a more just margin of profit. I admit, all the same, that the Australian Pharmaceutical Formulary "cannot replace the need for proper prescribing." The pharmacists have been indulgent towards the writer of prescriptions of the present day. Let them speak now. You, Sir, have provoked them. Yours, etc.,

A. C. F. HALFORD.

Brisbane, February 11, 1921.

[DR. HALFORD'S complaint in regard to the disapproval expressed in THE MEDICAL JOURNAL OF AUSTRALIA of ready-made formulæ appears to be based on the fact that the general practitioner is not in a position to study the British Pharmacopœia or the recent work in pharmacology, because he does not possess the book and because he does not read scientific articles in current literature. Is this an acceptable excuse?

Dr. Halford chides the JOURNAL for calling attention to a fact. Why is it unfair to state that pharmacists prescribe over the counter? Again, the excuse is not acceptable that some pharmacists prescribe because the medical practitioners are unwilling to or incapable of writing a prescription. One of the objects of the article was to call attention to the necessity of a better pharmacological equipment of the general practitioner.

Notwithstanding Dr. Halford's faith, we still maintain that certain—possibly only a few—proprietary preparations cannot be replaced by any prescription or ready-made formula. To quote the names of these preparations would be to give the manufacturers a very valuable gratuitous advertisement. THE MEDICAL JOURNAL OF AUSTRALIA cannot do this except through its "Analytical Department."

Does Dr. Halford approve of the advice contained in the Australian Pharmaceutical Formulary that certain prescriptions are indicated or recommended for special complaints? Is it a function of the pharmaceutical societies to teach the medical profession therapeutics?—EDITOR.]

ACCIDENTAL HÆMORRHAGE AND CÆSAREAN SECTION.

SIR: Dr. H. Cairns Lloyd, in his paper, writes: "There can be no question as to the inadvisability of this operation, for in a severe case the child is already dead and the condition of the uterus is such that evacuation of its contents would inevitably kill the mother from hæmorrhage." About two years ago I was called to a nursing home to see a patient. She was suffering from accidental hæmorrhage. She was pale and cold, with a stony hard uterus and continual pain. She had a small, running pulse and no sign of cervical dilatation. I at once ordered her into the King Edward Memorial Hospital, rang up the Honorary Surgeon, Dr. J. K. Couch, and told him her condition.

I anæsthetized and he performed Cæsarean section. The woman was off the table under one and a half hours from the time I first saw her. She did not "hæmorrhage," but made a good recovery. The only treatment she got before laparotomy was some morphia (one-quarter or one-half grain), when I had diagnosed her condition. She certainly had the appearance of a dying woman before the operation.

I have only seen four accidental hæmorrhages. Two, who were dilating, delivered themselves naturally and did well. A third I saw in the Riverina I delivered instrumentally after manual dilatation. She died from shock and hæmorrhage. The fourth case is the one I have described above.

As a general practitioner, I feel it almost a presumption to criticize Dr. Cairns Lloyd. I accept his obstetric opinion implicitly. Still, I feel sure that, had he seen the case reported, he would modify his views on the question of operative interference.

After my first fatal case I felt that accidental hæmorrhage without cervical dilatation must mean death. Since the successful Cæsarean section, I would certainly recommend it, as this is a complication where inactivity is surely not masterly. Yours, etc.,

M. KASNER MOSS.

Hay Street, Perth,
March 11, 1922.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered under the provisions of the *Medical Act of 1867* as duly qualified medical practitioners:

REED, ERIC BURTON, M.B., Ch.M., 1919 (Univ. Sydney), Bundaberg.
RICHARDSON, ARNOLD, M.B., B.S., 1921 (Univ. Sydney), Townsville.

VICTORIA.

THE undermentioned have been registered under the provisions of the *Medical Act, 1915*, as duly qualified medical practitioners:

BROAD, WILLIAM, M.B. *et* Ch.B., 1899 (Glas.), Hume Reservoir, *viâ* Albury, New South Wales.
COLLOPY, WILLIAM ANSELM, M.B., B.S., 1920 (Melb.), 7, Waltham Street, Richmond.
ORMOND-SMITH, LAURENCE, M.D., 1921 (Boston), Homœopathic Hospital, Melbourne.

Names of deceased practitioners removed from the Register:

EASTWOOD, FRANCIS HUDSON.
O'SHAUGHNESSY, MICHAEL.

Additional diploma registered:

TAIT, JOHN THOMSON, M.D., 1913, M.S., 1921 (Melb.).

Medical Appointments.

DR. T. M. MANSFIELD has been appointed Government Medical Officer at Ayr and a Health Officer under *The Health Acts, 1900 to 1917*, of Queensland.

* * * *

DR. H. W. SAVIGE (B.M.A.) has been appointed Government Medical Officer at Hughenden, Queensland.

* * * *

THE undermentioned have been appointed to constitute the Advisory Committee of the *Hospitals Act Amendment Act, 1921*, of South Australia: DR. F. S. HONE (B.M.A.), DR. W. T. HAYWARD, C.M.G. (B.M.A.), SIR J. C. VERCO (B.M.A.), DR. H. S. NEWLAND, D.S.O. (B.M.A.), DR. C. T. C. DE CRESPIGNY, D.S.O. (B.M.A.).

* * * *

THE undermentioned have been appointed members of the Fauna and Flora Board of South Australia: PROFESSOR F. WOOD JONES, DR. R. H. PULLEINE (B.M.A.).

* * * *

DR. F. N. LYNCH (B.M.A.) has been appointed Government Medical Officer at Urana, New South Wales.

* * * *

DR. H. C. ADAMS (B.M.A.) has been appointed to act as an Official Visitor to the Mental Hospital, Parramatta, and "Bay View House," Cook's River, in New South Wales, during the absence on leave of DR. SINCLAIR GILLIES.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx.

"LADY DAVIDSON" RED CROSS HOME, TURRAMURRA: Medical Superintendent.
LEONORA DISTRICT HOSPITAL, WESTERN AUSTRALIA: Resident Medical Officer.
NEGLECTED CHILDREN'S DEPÔT, ROYAL PARK, VICTORIA: Medical Officer and Superintendent.
ROYAL NORTH SHORE HOSPITAL OF SYDNEY: Honorary Assistant Pathologist (Further Applications).

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C..

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney	Australian Natives' Association Ashfield and District Friendly Societies' Dispensary Balmain United Friendly Societies' Dispensary Friendly Society Lodges at Casino Leichhardt and Petersham Dispensary Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney Marrickville United Friendly Societies' Dispensary North Sydney United Friendly Societies People's Prudential Benefit Society Phœnix Mutual Provident Society
VICTORIA: Honorary Secretary, Medical Society Hall, East Melbourne	All Institutes or Medical Dispensaries Australian Prudential Association Proprietary, Limited Manchester Unity Independent Order of Oddfellows Mutual National Provident Club National Provident Association
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane	Brisbane United Friendly Society Institute Hampden District Hospital Stannary Hills Hospital
SOUTH AUSTRALIA: Honorary Secretary, 3, North Terrace, Adelaide	Contract Practice Appointments at Renmark Contract Practice Appointments in South Australia
WESTERN AUSTRALIA: Honorary Secretary, 6, Bank of New South Wales Chambers, St. George's Terrace, Perth	All Contract Practice Appointments in Western Australia
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington	Friendly Society Lodges, Wellington, New Zealand

Diary for the Month.

MAR. 28.—New South Wales Branch, B.M.A.: Council.
MAR. 29.—Victorian Branch, B.M.A.: Council.
MAR. 30.—South Australian Branch, B.M.A.: Branch.
MAR. 31.—New South Wales Branch, B.M.A.: Annual Meeting.
APR. 4.—New South Wales Branch, B.M.A.: Council.
APR. 5.—Victorian Branch, B.M.A.: Branch.
APR. 7.—Queensland Branch, B.M.A.: Branch.
APR. 11.—New South Wales Branch, B.M.A.: Ethics Committee.
APR. 12.—Western Australian Branch, B.M.A.: Council.
APR. 12.—Melbourne Pædiatric Society.
APR. 13.—Victorian Branch, B.M.A.: Council.
APR. 13.—Brisbane Hospital Clinical Society: Meeting.
APR. 14.—Queensland Branch, B.M.A.: Council.
APR. 14.—South Australian Branch, B.M.A.: Council.
APR. 15.—Northern Suburbs Medical Association, New South Wales.
APR. 18.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
APR. 19.—Western Australian Branch, B.M.A.: Branch.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, B.M.A. Building, 30-34, Elizabeth Street, Sydney. (Telephone: B. 4635.)